A Study on

CLINICAL PROFILE AND SHORT TERM FOLLOW UP OF ACUTE CHOLECYSTITIS

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CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. S. KARTHIKEYAN** on a study of **CLINICAL PROFILE AND SHORT TERM FOLLOW UP OF ACUTE CHOLECYSTITIS** during his **M.S. (General Surgery)** course from 2003 – 2006 at Kilpauk Medical College and Hospital, Chennai – 600 010 under my guidance and supervision.

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CONTENTS

1. INTRODUCTION

Biliary tract diseases are very common in developed countries. But there is recent trend of increase in incidence in developing countries which can be attributable due to the westernization of dietary habits. In India due to fast emerging western culture and mushrooming of number of western fast food restaurants, obesity and gall stone maladies are becoming more prevalent. Prevalence of the disease in India [14] in males is 4% and females 8% with the prevalence being more in northern India than the south.

The operations on gall bladder and biliary tree ranks next to hernia surgery and appendicectomy. The research in the pathogenesis of the gall stone disease during the past two decades have revolutionized the treatment modalities including surgery. Proliferating research in the minimally invasive surgery, especially on laparoscopy and endoscopy has changed the management approaches in the biliary tract surgeries.

With the advent of newer imaging investigations like CT cholangiography and Magnetic Resonance Cholangio Pancreaticography, the intervention could be done at an early stage, thereby reducing the mortality and morbidity.

2. AIM OF SUTDY

This study was made with the following intentions:

i. To analyze about the etiological factors, presentation, predisposing conditions, diagnosis and treatment options in consecutive cases of acute cholecystitis.

ii. To compare the various diagnostic modalities and treatment options.

iii. To analyze the surgical outcome in a given patient.

iv. To recognize the postoperative complications in a short term follow up.

HISTORICAL PRESPECTIVE[1]

1) Gall bladder has been mentioned as early as 400 B.C. Hippocrates who noticed that hardening of the liver was bad sign in jaundice, but persistent jaundice may be due to cancer or cirrhosis of liver both of which are marked by hardening of the liver tissue and enlargement of the organ.

2) Gall stones have been found in Egyptician mummies since 2nd and 3rd AD. Plymy of Rome demonstrated double gall bladder.

3) Soranus of Ephesus described the triad of extra hepatic obstructive jaundice i.e. jaundice, achouluric stools, dark urine and itching.

4) 500 A.D - Alexander Trallia described Bililary Stones

5) 1809 to 1826 - Berzelieus and Gmelin shown bile acid existence

6) 1843 - Lieberg coined the term bile acid

7) 1844 - Bovelius and Hoffmann and Hoppe-Saylor postulated enterohepatic circulation confirmed by weiss.

HISTORY OF SURGERY ON GALL BLADDER

1) 1667 - Stalport reported evacuation of gall bladder stones on opening of abdominal abcesses.

2) 1867 - John Bobbs performed 1st cholecystectomy and stone removal.

3) 1882 - Carel Johnon Langenback performed 1st cholecystectomy in USA.

4) Sims, Kocher, Keen , Tait and Moyniham pioneered gall bladder surgery later.

5) Since the 1st laproscopic cholecystectomy by Mouret, Lyon of France it has been approach for gall stone disease.

HISTORY OF IMAGING OF GALL BLADDER[4,18]

1) 1924 - Graham and Cole described oral cholecystography.

Pyrioxylidine compounds have been replaced by Imino acetic acid compound.

2) 1931 - Per-operative cholangiogram described by Mirrizi.

3) 1980's - ERCP was described by Carlsen and Dembling.

EMBROYLOGY

DEVELOPMENT OF THE LIVER AND BILIARY TRACT[7]

(Fig1.1)

The human liver is formed from two primordia: the liver diverticulum and the septum transversum. Close proximity of cardiac mesoderm,. The liver diverticulum forms through proliferation of endodermal cells at the cranioventral junction of the yolk sac with the foregut and grows into the septum transversum in a cranioventral direction. The endodermal cells differentiate into hepatocytes and epithelial cells of the bile ducts. Hepatic portion differentiates into proliferating cords of hepatocytes and the intrahepatic bile ducts. The cystic portion, which initially is a cord of epithelial cells, forms the gallbladder, common duct, and cystic duct through a process of elongation and recanalization.

A ring of hepatocytes in close proximity to the portal vein branches first transforms into bile duct–type cells. This double-walled cylinder with a slitlike lumen, the ductal plate, can be detected at 9 weeks of gestation. Thus, the entire network of interlobular and intralobular bile ductules develops from the limiting plate.

The gallbladder and extrahepatic bile ducts start to develop from hepatic endodermal cells and hepatoblasts immediately after formation of the liver primordium.The original hepatic diverticulum differentiates cranially into proliferating hepatic cords and bile ducts and caudally into the gallbladder.

SURGICAL ANATOMY

BILE DUCTS[7, 9] (Fig.1.2)

A general feature of bile ductules is their anatomic intimacy with portal blood and lymph vessels.

Bile enters the small terminal bile ductules (the canals of Hering), which have a basement membrane and are lined by three to six ductal epithelial cells. The canals of Hering provide a conduit through which bile may traverse to enter the larger perilobular or intralobular ducts. The interlobular bile ducts form a anastomosing network that closely surrounds the branches of the portal vein.

The common hepatic duct emerges from the porta hepatis after the union of the right and left hepatic ducts, each of which is 0.5 to 2.5 cm long. The confluence of the right and left hepatic ducts is outside the liver in approximately 95% of cases.

In the adult, the common hepatic duct is approximately 3 cm long and is joined by the cystic duct, usually at its right side, to form the common bile duct.

The cystic, common hepatic, and common bile ducts possess mucosa, submucosa, and muscularis. The ducts are lined by a single layer of columnar epithelium. Mucus secreting tubular glands can be found at regular intervals in the submucosa, with openings to the surface of the mucosa.

The common bile duct is approximately 7 cm long, runs between layers of the lesser omentum, and lies anterior to the portal vein and to the right of the hepatic artery. The common bile duct normally is approximately 0.5 to 1.5 cm in diameter. The wall of the extrahepatic bile ducts is supported by a layer of connective tissue with an admixture of occasional smooth muscle fibers. The smooth muscle component is conspicuous only at the neck of the gallbladder and at the lower end of the common duct. The common bile duct passes retroperitoneally behind the first portion of the duodenum in a notch on the back of the head of the pancreas and enters the second part of the duodenum. The duct then passes obliquely through the posterior medial aspect of the duodenal wall and joins the main pancreatic duct to form the ampulla of Vater .The mucus membrane bulge produced by the ampulla forms an eminence, the duodenal papilla. (Fig.1.2)

As they course through the duodenal wall, the bile and pancreatic ducts are invested by a thickening of both the longitudinal and circular layers of smooth muscle of the sphincter of Oddi. It is usually composed of several parts: (1) the sphincter choledochus, which consists of circular muscle fibers that surround the intramural portion of the common duct immediately before its junction with the pancreatic duct; (2) a pancreatic sphincter, which is present in approximately one third of individuals and surrounds the intraduodenal portion of the pancreatic duct before its juncture with the ampulla; (3) the fasciculi longitudinales, which are composed of longitudinal muscle bundles that span

intervals between the bile and pancreatic ducts; and (4) the sphincter ampullae, which consists of longitudinal muscle fibers that surround a sparse layer of circular fibers around the ampulla of Vater.

The arterial supply of the bile ducts arises mainly from the right hepatic artery. An extraordinarily rich plexus of capillaries surrounds bile ducts as they pass through the portal tracts. Blood flowing through this peribiliary plexus empties into the hepatic sinusoids via the interlobular branches of the portal vein.

An abundant anastomotic network of blood vessels from branches of the hepatic and gastroduodenal arteries supplies the common bile duct. The supraduodenal portion of the duct is supplied by vessels running along its wall inferiorly from the retroduodenal artery and superiorly from the right hepatic artery. Injury to these blood vessels can result in bile duct stricturing.

The lymphatic vessels of the hepatic, cystic, and proximal portions of the common bile duct empty into glands at the hilum of the liver.

GALL BLADDER [7,9]

The gallbladder is a storage reservoir for, bile acids and on the undersurface of the right lobe of the liver. This distensible pear-shaped structure is 3 cm wide and 7 cm long in the adult and has a capacity of 30 to 50 mL. The gallbladder is covered anteriorly by an adventitia that is fused with the

capsule of the liver. On its posterior aspect and at the apex, it is covered by the visceral peritoneum. The anterior portion of the fundus is located at the level of the right lateral border of the musculus rectus abdominis and the ninth costal cartilage. The posterior aspects of the fundus and body lie close to the transverse colon and duodenum, respectively. The infundibulum is an area of tapering between the gallbladder body and the neck. Hartmann's pouch is a bulging of the inferior surface of the infundibulum that lies close to the neck of the gallbladder.

The gallbladder is connected at its neck to the cystic duct, which empties into the common bile duct. The cystic duct is approximately 4 cm long and maintains continuity with the surface columnar epithelium, lamina propria, muscularis, and serosa of the gallbladder. The mucous membrane of the gallbladder neck forms the spiral valve of Heister, that is involved in regulating flow into and out of the gallbladder.

The gallbladder is supplied by the cystic artery, which usually arises from the right hepatic artery. The artery divides into two branches near the neck of the gallbladder: a superficial branch that supplies the serosal surface and a deep branch that supplies the interior layers of the gallbladder wall. However, variations in the origin and course of the cystic artery are common. Because the cystic artery is an end artery, the gallbladder is particularly susceptible to ischemic injury and necrosis that result from inflammation or interruption of hepatic arterial flow.

The cystic vein provides venous drainage from the gallbladder and cystic ducts and commonly empties into the portal vein and occasionally directly into the hepatic sinusoids.

The lymph vessels of the gallbladder are connected with the lymph vessels of Glisson's capsule. Subserous and submucosal lymphatics empty into a lymph gland near the neck of the gallbladder.

The sympathetic innervation of the gallbladder originates from the celiac axis. Visceral pain is conducted through sympathetic fibers and is frequently referred to the right subcostal, epigastric, and right scapular regions. Branches of vagi provide parasympathetic innervation contributes to the regulation of gallbladder motility.

CHOLECYSTOHEPATIC TRIANGLE

Calot (1891) described its importance. It is formed by cystic duct and gall baaldder laterally, right lobe of liver above, and the common hepatic duct medially. Its contents include

- Right hepatic artery which enter either posterior(87%) or anterior(13%).
- Aberrant rt. Hepatic artery from superior mesenteric passes through it.
- Cystic artery arises from either one of this, and divides into superficial and deep branches
- Duplication of cystic artery seen in 25 % if cases.
- Aberrant / accessory hepatic duct that enter either cyctic or common hepatic duct in 15% of cases.

PHYSIOLOGY AND FUNCTIONS OF BILE[6,13]

Bile formation is essential for normal intestinal lipid digestion and absorption, cholesterol homeostasis, and the excretion of lipid-soluble xenobiotics, drugs, and heavy metals.

Bile is a complex lipid-rich micellar solution that is isoosmotic with plasma and composed primarily of water, inorganic electrolytes, and organic solutes such as bile acids, phospholipids , cholesterol, and bile pigments. The volume of hepatic bile secretion is estimated to be between 500 and 600 mL per day.

Bile acids are essential for intestinal absorption of cholesterol and fat-soluble vitamins and play an important role in digestion of dietary fats. Bile acids promote intestinal absorption by solubilizing dietary lipids and their digestion products as mixed micelles to facilitate their aqueous diffusion across the intestinal mucosa. Fat-soluble vitamins (A, D, E, and K1) are not absorbed in the absence of bile acid micelles, and disturbances in the secretion or enterohepatic cycling of bile acids may lead to fat-soluble vitamin deficiency. Bile acids are synthesized from cholesterol in the pericentral hepatocytes

COMPOSITION OF HEPATIC BILE

COMPONENT	CONCENTRATION (mMol/l)
Electrolytes	
Na^+	141-165
K^+	2.7-6.7
Cl^-	77-117
HCO_3-	12-55
Ca^{2+}	2.5-6.5
Mg^{2+}	1.5-3.0
Organic anions	
Bile acids	3-45
Bilirubin	1-2
Lipids	
Lecithin	140-810 (mg/dL)
Cholesterol	97-320 (mg/dL)
Protein	2-20(mg/mL)
Peptides and amino acids	
Glutathione	3-5
Glutamate	0.8-2.5
Aspartate	0.4-1.1
Glycine	0.6-2.6

THE ENTEROHEPATIC CIRCULATION (Fig. 1.3)

The anatomic components of the enterohepatic circulation are the liver, biliary tract, intestine, portal venous circulation, and, to a lesser extent, the colon, systemic circulation, and kidney.

During fasting, bile acids traverse the biliary tract and are concentrated approximately 10-fold in the gallbladder.

During the digestion of a large meal, the gallbladder remains contracted, and bile acids secreted by the liver bypass the gallbladder and pass directly into the duodenum. During the digestion of a large meal, the gallbladder remains

contracted, and bile acids secreted by the liver bypass the gallbladder and pass directly into the duodenum. Less than 10% of the intestinal bile acids eliminated in the faeces.

In the small intestine, bile acids are absorbed predominantly by an active transport system restricted to the terminal ileum.

The intestine may reabsorb between 10 and 30 g of bile acids per day. Approximately 0.2 to 0.6 g of bile acids escape re-absorption and are eliminated in the stool each day.

DEFINITIONS

CHOLECYCTITIS **(It literally means inflammation of gall bladder)**

BILIARY COLIC AND CHRONIC CHOLECYSTITIS[4,5]

Biliary colic is the most common symptom of cholelithiasis. Approximately 75% of symptomatic patients with gallstone disease seek medical attention as a result of episodic abdominal pain. The pain of biliary colic is visceral in origin and therefore is poorly localized, the patient experiences episodes of upper abdominal pain, usually in the epigastrium or right upper quadrant but sometimes in other abdominal locations. The pain may be precipitated by eating a meal, but more commonly there is no inciting event; pain may even begin during sleep.

Chronic cholecyctitis is caused recurrent episodes of acute cholecystitis.

ACUTE CHOLECYSTITIS[4,5]

Acute cholecystitis is considered the most frequent complication of gallstone disease. Inflammation of the gallbladder wall that is associated with a clinical picture of abdominal pain, right upper quadrant tenderness, fever, and leukocytosis is the hallmark of acute cholecystitis. In 90% of acute cholecystitis, the cause is a gallstone that obstructs the cystic duct. In 10% of cases, cholecystitis occurs in the absence of gallstones and is termed *acute acalculous cholecystitis.* Although it is classical, it is inappropriate to divide cholecyctitis in acute and chronic forms as they are part of spectrum of same disease.

There are certain uncommon conditions affecting the gall bladder in which there is chronic inflammatory changes in the wall of gall bladder

They are called cholecystoses.[2]

1. CHOLESTEROSIS *(straw berry gall bladder)*

Submucous aggregation of cholesterol crystal and cholesterol esters .

2. CHOLESTEROL POLYPOSIS OF GALL BLADDER

3. CHOLECYCTITIS GLANDULARIS PROLIFERENS (Polyp, adenomyomatosis and intramural diverticulosis)

4. DIVERTICULOSIS OF GALL BLADDER

It usually manifest as black pigment stones impacted in the out pouchings of lacunae of Luschka. Diverticulosis is demonstrated by cholecystography.

EPIDEMIOLOGY, RISK FACTORS, PATHOGENESIS, AND NATURAL HISTORY OF GALLSTONES

PREVALENCE AND INCIDENCE[4,14]

There are several large studies, the older studies have taken into account the necropsy data. But the newer ones are using the ultrasonographic data. Several interesting points can be derived from these data. In general, gallstones are approximately two times more common in women than in men, and at least 10% of the general populations have gallstones.

Most series indicate that the prevalence for women between the ages of 20 and 55 varies from 5% to 20% and is 25% to 30% after the age of 50. The prevalence for men is approximately half that for women in a given age group.

ETHNIC PREDISPOSITION[14,19]

Genetic factors play a key role in the pathogenesis of gallstone disease. These are likely to be multifactorial and to vary among populations. Within a given population, in first-degree relatives of index cases with gallstone disease, gallstones are 4.5 times more likely to develop

than in age- and gender-matched controls, implying a strong genetic influence

TRUE INCIDENCE

The largest study to date is of the Danish population and was published in 1991.

The 5-year incidence of gallstones in men aged 30, 40, 50, and 60 years was 0.3%, 2.9%, 2.5%, and 3.3%, respectively. The corresponding rates for women were 1.4%, 3.6%, 3.1% and 3.7%. Women clearly had a higher incidence than men at 30 and 40 years of age, but this difference disappeared with increasing age.

GALL STONE MORPHOLOGY AND COMPOSITION [2,3,10]

Gallstones are categorized as cholesterol, black pigment, or brown pigment stones, largely on the basis of their composition.

Cholesterol stones, the most common type of gallstones, are composed purely of cholesterol or have cholesterol as the major chemical constituent. These stones can often be identified by inspection. Microscopically, pure cholesterol stones are composed of many long, thin cholesterol monohydrate crystals bound together by a matrix of mucin glycoproteins with a black core composed of a calcium salt of

unconjugated bilirubin. Mixed cholesterol gallstones consist of more than 50% cholesterol and are slightly more common than pure cholesterol stones.

Black pigment stones are composed of either pure calcium bilirubinate or polymer-like complexes with calcium, copper, and large amounts of mucin glycoproteins.They are common in patients with cirrhosis and chronic hemolytic states.

Brown pigment stones are composed of calcium salts of unconjugated bilirubin, with varying amounts of cholesterol and protein. These stones are usually associated with infection. Bacteria present in the biliary system produce ß-glucuronidases that form calcium salts of unconjugated bilirubin, deconjugated bile acids, and saturated long-chain fatty acids. Microscopically, brown stones contain cytoskeletons of bacteria, which is consistent

TYPE	MIXED	PURE CHOLESTEROL	BLACK PIGMENT	BROWN PIGMENT
COMPOSITION	**Cholesterol / Calcium salts**	**Cholesterol**	**Pigment polymer/calcium bilirubinate**	**Calcium bilirubinate/calcium salts of fatty acids.**
SHAPE	**Round**	**Round/ smooth**	**Spiky/ faceted**	**Ovoid / Irregular**
COLOUR	**Brown and yellow specks or rings**	**Yellow to white pigment in center**	**Black and shiny or dull**	**Brown soft and earthy**
MICROBES	**Sterile**	**Sterile**	**Sterile**	**Infected**

RISK FACTORS	**Female/ obesity**	**Female/ obesity**	**Hemolytic disorders**	**Cholangitis**

ETIOLOGY, RISK FACTORS AND CO-MORBID CONDITIONS

Within a population, gallstones occur sporadically but not randomly.

AGE AND GENDER

Because gallstones rarely dissolve spontaneously, the cumulative prevalence of gallstones increases with age[2,6]

Gender is a prominent risk factor for gallstone formation: most studies report a two- to threefold higher risk in women than in men. The increased incidence in women is present through the fifth decade, after which the incidence rates in men and women become essentially equal.

OBESITY, WEIGHT LOSS, AND TOTAL PARENTERAL NUTRITION

Obesity is a well-known risk factor for cholelithiasis. A large prospective study of obese women found a strong linear association between body mass index (expressed in kilograms per square meter [kg/m^2]) and the reported incidence of cholelithiasis. In this study, those with the highest body

mass index (greater than 45 kg/m^2) had a seven-fold increased risk of development of gallstones compared to non obese controls. The same applies to men but association is weak[2,4,5]

Rapid weight loss is a recognized risk factor for cholesterol gallstone formation. Gallstones develop in approximately 25% of obese patients who are on strict dietary restriction.

Hepatic cholesterol secretion increases during caloric restriction. Additional factors may include increased production of mucin (a potent stimulator of cholesterol crystal nucleation) and decreased gallbladder motility.

Total parenteral nutrition (TPN) is associated with the development of acalculous cholecystitis as well as cholelithiasis and cholecystitis[2,5]

In as many as 45% of adults and 43% of children, gallstones develop after 3 to 4 months of TPN

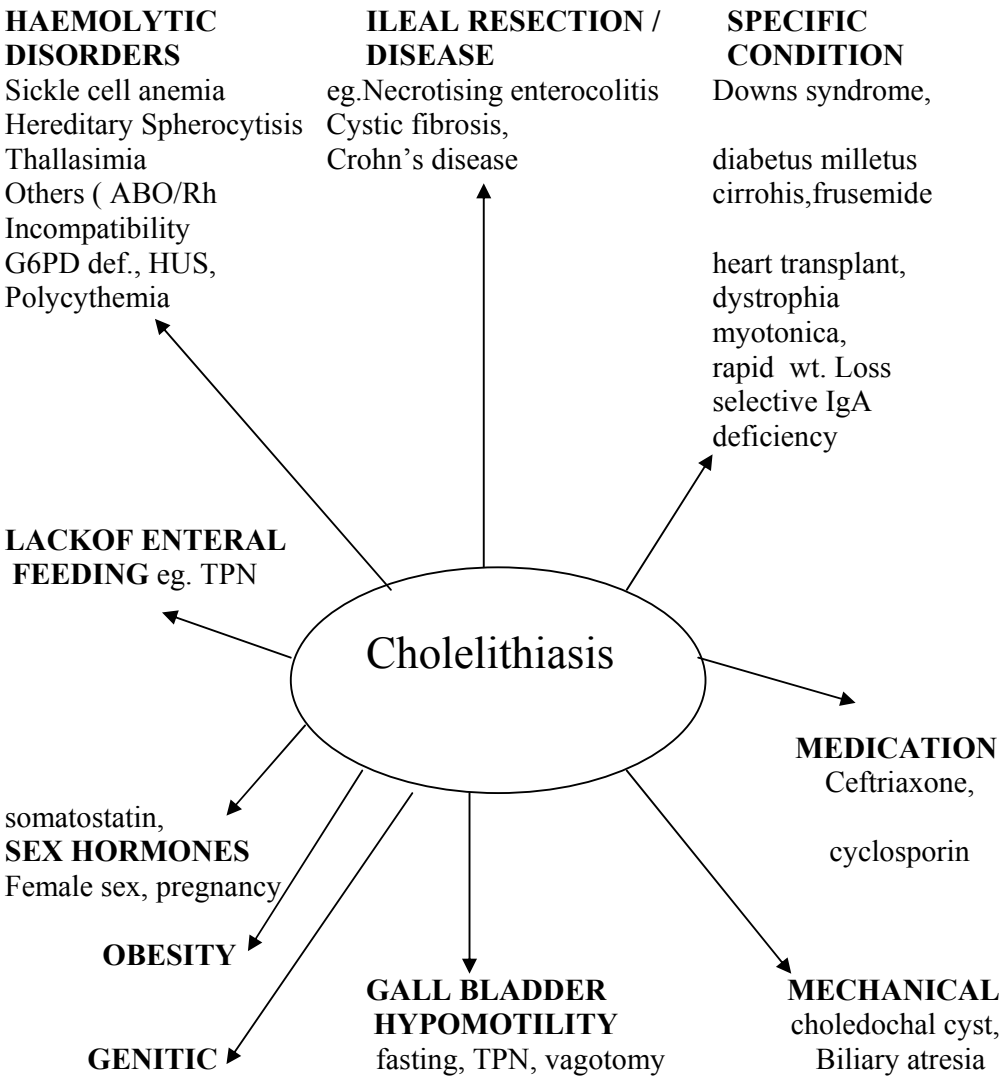
The primary physiologic defect is gallbladder hypomotility with bile stasis, which results from prolonged fasting. In addition, if there is failure of the sphincter of Oddi to relax.

PREGNANCY AND PARITY[22,8]

Pregnancy is a clear risk factor for the development of biliary sludge and gallstones. Bile becomes more lithogenic during pregnancy, possibly as a result of increased estrogen levels, which result in increased

cholesterol secretion and supersaturation of bile. In addition, gallbladder volume doubles and stasis develops. Increased progesterone levels impair gallbladder motility.

ETIOLOGY



DRUGS

Estrogen is the most extensively studied. Exogenous estrogen increases biliary cholesterol secretion.[4,22]

Lipid lowering drugs as a class alter key pathways in cholesterol and bile acid synthesis and metabolism. Clofibrate has the greatest association with increased gallstone formation The drug induces cholesterol supersaturation in bile and diminishes bile acid concentrations by reducing the activity of cholesterol 7-α-hydroxylase.

Octreotide, a somatostatin analog, increases the incidence of gallstones in patients who are treated with the drug for acromegaly.

DIET AND LIPID PROFILE[27]

A high serum cholesterol level does not seem to be a risk factor for development of gallstones. On the other hand, hypertriglyceridemia is positively associated with an increased incidence of gallstones. HDL cholesterol levels are inversely correlated with the presence of gallstones [27]

The ingestion of refined sugars and decreased physical activity are both positively associated with the presence of gallstones

SYSTEMIC DISEASE

Diabetics are more prone than nondiabetics to the development of complications associated with cholelithiasis. Diabetes is also associated

with hypertriglyceridemia, obesity, and gallbladder hypomotility, known risk factors for gallstone formation [23]

Diseases of the ileum are recognized risk factors for the development of gallstones. Crohn's disease is the most common systemic illness that affects the terminal ileum. Patients with Crohn's disease have a two- to threefold increased risk of formation of gallstones.

Spinal cord injuries are associated with a high prevalence of gallstones. Moonka and associates reported a prevalence of gallstones of 31% .

PATHOGENESIS

CHOLESTEROL SUPERSATURATION [4,19] (Fig.1.6)

Cholesterol is essentially insoluble in water and therefore relies on the detergent activity of bile salts and the polar phospholipids (lecithin) to stay in solution. The degree of cholesterol saturation in gallbladder bile is the most important single determinant of crystal formation in humans.

The liver is the only organ that can permanently eliminate cholesterol from the body.

Rates of cholesterol esterification may influence the lithogenicity of bile because drugs that reduce esterification (e.g. progesterone and clofibrate) tend to increase the secretion of cholesterol.

Bile salts, which are the most abundant solutes in bile, are critical in determining cholesterol solubilization. Only two primary bile acids are synthesized in humans: cholate and chenodeoxycholate; each represents approximately 35% of the total bile acid pool. The secondary bile acids, deoxycholic acid and lithocholic acid represent approximately 24% and 1% to 3%, of the pool, respectively. Ursodeoxycholic acid is a tertiary bile acid that contributes up to 4% of the bile acid pool. All bile acids are conjugated with glycine or taurine in approximately a 2:1 ratio prior to secretion

The more hydrophobic the bile acid, the greater is its ability to induce cholesterol secretion and suppress bile acid synthesis. The combination of increased cholesterol secretion and decreased bile acid synthesis leads to more lithogenic bile. Prostaglandins stimulate secretion of mucin, a proposed pronucleator thus may contribute to the formation of lithogenic bile.

NUCLEATING AND ANTINUCLEATING FACTORS[19]

Supersaturated bile, the first step in gallstone formation is nucleation: the condensation or aggregation process by which a propagable submicroscopic crystal or amorphous particle is formed from supersaturated bile. After nucleation, crystallization occurs, producing cholesterol monohydrate crystals that can agglomerate to form

macroscopic gallstones. Mucin glycoproteins bind to cholesterol, phospholipids, and bilirubin.

The binding of cholesterol rich vesicles to the hydrophobic regions seems to mediate the observed accelerated nucleation. Because mucin and bilirubin are frequently found in the core of cholesterol gallstones, this complex may serve as a nidus for stone formation. Mucin secretion is excessive in lithogenic bile.

Pronucleators h isolated in model bile systems by lecithin chromatography using concanavilin A sepharose. These pronucleators include immunoglobulin G (IgG) and IgM, aminopeptidase N, haptoglobin, and α1-acid glycoprotein.

Antinucleating proteins that have been identified in model bile systems include apolipoproteins A-I and A-II and a biliary glycoprotein.

Biliary calcium concentration plays a role in bilirubin precipitation and gallstone formation because calcium salts are present in most cholesterol gallstones.

GALL BLADDER HYPOMOTILITY[4,23]

The contribution of the gallbladder to the pathogenesis of gallstones is widely recognized. The mucosa of the gallbladder has one of the highest rates of water absorption in the body. The volume of bile residing in the

gallbladder decreases by 80% to 90% as a result of active sodium transport coupled with passive water absorption.

Neural control of gallbladder emptying is mediated by both parasympathetic and sympathetic innervation. Inhibiting the cholinergic input with atropine increases fasting volumes and reduces emptying after meals in response to cholecystokinin (CCK).

The stimulants of CCK release are, in order of decreasing potency, long-chain fatty acids, amino acids, and carbohydrates.

EPIDEMIOLOGY OF PIGMENT STONES[10, 20, 27]

Pigment stones account for 10% to 25% of all gallstones

Black pigment stones form in the gallbladder as a result of increased production of unconjugated bilirubin, which then precipitates as calcium bilirubinate to form stones. Therefore, black pigment stone formation is typically associated with chronic hemolysis (e.g., β-thalassemia, hereditary spherocytosis, sickle cell hemoglobinopathy), cirrhosis, and pancreatitis.

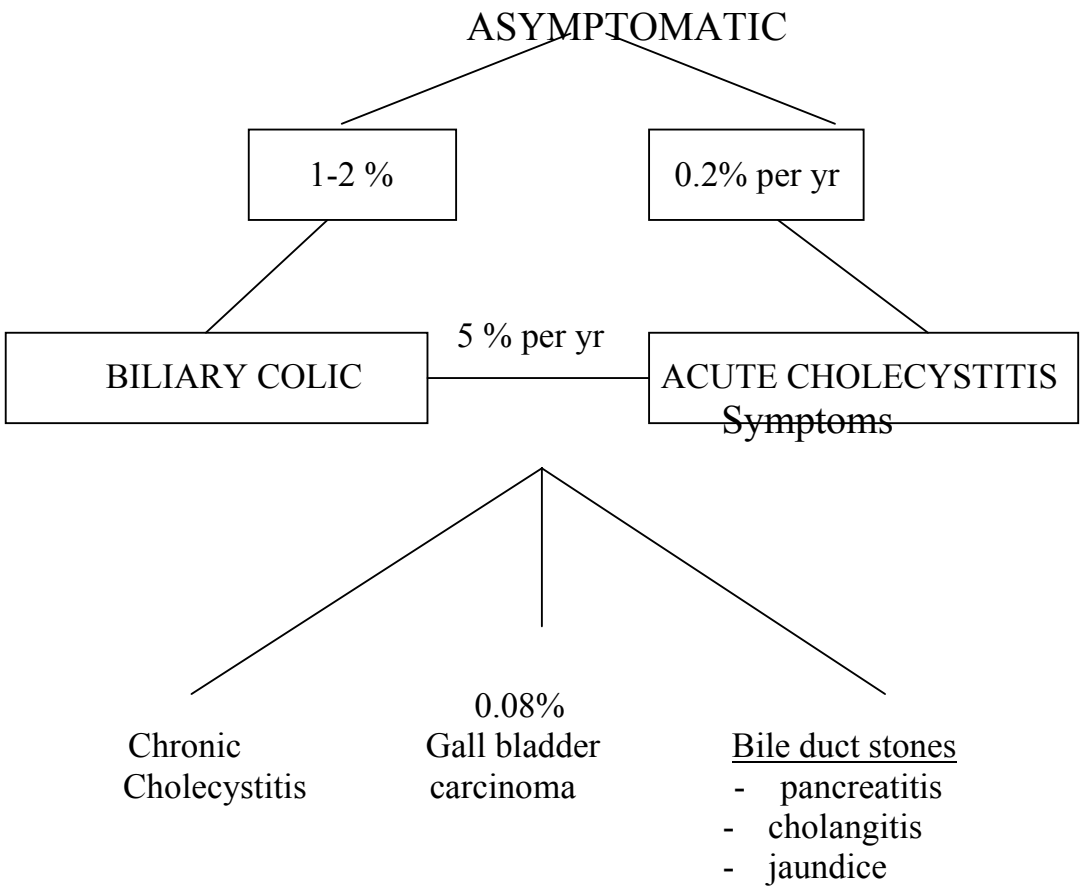
Brown pigment stones are more common in areas where biliary infections are more prevalent. They can occur in the gallbladder or in the biliary tree.

PATHOGENESIS OF PIGMENT STONES (Fig 1.4)

Black pigment stones are composed primarily of calcium bilirubinate but also contain calcium carbonate and calcium phosphate. The unifying characteristic of the conditions that predispose to black stone formation is the hypersecretion of bilirubin conjugates (especially monoglucuronides) into the bile. In presence of hemolysis output of bilirubin conjugates increases 10-fold.

An acidification defect has also been documented, this buffering effect facilitates the supersaturation of calcium carbonate and phosphate, which would not occur at a more acidic pH, and allows precipitation.

NATURAL HISTORY OF GALL STONES[2,4,21]



ASYMPTOMATIC STONES[21]

The Group for Epidemiology and Prevention of Cholelithiasis (GREPCO) in Rome followed the natural history of 151 subjects with gallstones, The cumulative rate of biliary complications was 3% at 10 years

SYMPTOMATIC STONES

The natural history of symptomatic gallstones follows a more aggressive course. The National Cooperative Gallstone Study showed that of those patients who had an episode of uncomplicated biliary pain in the year before entering the study, 38% per year had recurrent biliary pain. The risk of development of biliary complications is estimated to be 1% to 2% per year and is thought to remain relatively constant over time.

DIABETES MELLITUS[23, 5]

Diabetic patients with incidental cholelithiasis were long considered to have an increased risk of serious complications even though the gallstones were asymptomatic, study showed that after 5 years of follow-up, symptoms developed in 15%.

CLINICAL MANIFESTATIONS OF GALLSTONE DISEASE [1, 4, 17] (Fig. 1.5, Table (a))

A gallstone can cause symptoms by only two mechanisms: it can obstruct the cystic duct or common bile duct, or, much more rarely, it can erode through the gallbladder wall.

It is emphasized that most gallstones never cause symptoms, and the purely incidental discovery of cholelithiasis rarely warrants specific intervention. Possible exceptions to this general dictum include the following:

1. A young patient with sickle cell anemia and incidental cholelithiasis in whom an abdominal pain crisis would be difficult to distinguish from biliary colic or acute cholecystitis.
2. A young woman of American Indian ancestry with incidental cholelithiasis in whom prophylactic cholecystectomy may be warranted to prevent the delayed complication of gallbladder cancer.
3. Any patient with gallbladder wall calcification (porcelain gallbladder) who is an acceptable surgical risk for the purpose of preventing gallbladder carcinoma as a late complication
4. A patient with incidental cholelithiasis who is planning prolonged space travel or other extremely remote assignments

COMMON COMPLICATIONS

BILIARY COLIC AND CHRONIC CHOLECYSTITIS

Biliary colic is the most common symptom of cholelithiasis. Approximately 75% of symptomatic patients with gallstone disease seek medical attention as a result of episodic abdominal pain

PATHOGENESIS[19, 21]

The syndrome of biliary colic is caused by intermittent obstruction of the cystic duct by one or more gallstones. It is not necessary that inflammation of the gallbladder accompany the obstruction—only that symptoms be caused by it

The most common histologic changes observed are mild fibrosis of the gallbladder wall with a round cell infiltration and an intact mucosa. However, recurrent episodes of biliary colic may be associated with a scarred, shrunken gallbladder and Rokitansky-Aschoff sinuses (intramural diverticula). Bacteria can be cultured from gallbladder bile or gallstones themselves in approximately 10% of patients.

CLINICAL MANIFESTATIONS

The pain of biliary colic is visceral in origin and therefore is poorly localized. Patient experiences episodes of upper abdominal pain, usually in the epigastrium or right upper quadrant but sometimes in other abdominal locations.

The pain of biliary colic is steady rather than intermittent as would be suggested by the word *colic*. The pain gradually increases over a period of 15 minutes to 1 hour and then remains at a plateau for 1 hour or more before slowly resolving.

In order of decreasing frequency, the pain is felt maximally in the epigastrium, right upper quadrant, left upper quadrant, and various parts of the precordium or lower abdomen. Diaphoresis and nausea with some vomiting are common.

Physical examination findings are usually normal with only mild to moderate gallbladder tenderness during an attack and perhaps mild residual tenderness that lasts several days after an attack.

DIAGNOSIS

In a patient with uncomplicated biliary colic, results of laboratory studies are usually completely normal. In general, the first (and in most cases only) imaging study to be performed on patients with biliary colic is an ultrasonographic examination of the right upper quadrant.

OCG is reserved for patients in whom medical dissolution therapy or lithotripsy of the gallstones is planned.

The Meltzer-Lyon test now has had a modest resurgence in popularity because of the ease with which bile can be obtained at the time of upper endoscopy or ERCP.

DIFFERENTIAL DIAGNOSIS

The most common diseases to a patient with recurrent, episodic upper abdominal symptoms are reflux esophagitis, peptic ulcer, pancreatitis, renal colic, colonic disorders such as diverticulitis and carcinoma, radiculopathy, and angina pectoris. Careful history taking is critical in sorting out the differential diagnosis of recurrent upper abdominal pain. irritable bowel syndrome, like biliary colic, is common in young women, but abdominal pain in this disorder has a distinct relationship with bowel movements

ACUTE CHOLECYSTITIS [1,3,4,17,25]

Acute cholecystitis is considered the most frequent complication of gallstone disease. Inflammation of the gallbladder wall that is associated with a clinical picture of abdominal pain, right upper quadrant tenderness, fever, and leukocytosis is the hallmark of acute cholecystitis.

In 10% of cases, cholecystitis occurs in the absence of gallstones and is termed *acute acalculous cholecystitis.* Acute acalculous cholecystitis is

more common in critically ill elderly men and is associated with high morbidity and mortality rates.

PATHOGENESIS

Acute cholecystitis generally occurs when a stone becomes impacted in the cystic duct and causes chronic obstruction. Stasis of bile within the gallbladder lumen results in damage of the gallbladder mucosa with consequent release of intracellular enzymes and activation of a cascade of inflammatory mediators. Enteric bacteria can be cultured from gallbladder bile in approximately one half of patients with acute cholecystitis.

PATHOLOGY

If the gallbladder is examined in the initial days of an attack of acute cholecystitis, distention is usually noted, with impaction of a stone in the cystic duct. On opening of the gallbladder, inflammatory exudate and, rarely, pus are present. Later in the attack, bile pigments that are normally present have been absorbed and replaced by thin mucoid fluid, pus, or blood. If the attack of acute cholecystitis is untreated for a long period and the cystic duct remains obstructed, the lumen of the gallbladder may become distended with clear mucoid fluid, so-called hydrops of the gallbladder.

Histologic changes range from mild acute inflammation with edema to necrosis and perforation of the gallbladder wall. When the gallbladder is resected for acute cholecystitis and no stones are found, the specimen should be carefully examined histologically for evidence of vasculitis or cholesterol emboli, because these systemic disorders may be manifested by acalculous cholecystitis.

CLINICAL MANIFESTATIONS

The pain of biliary colic if present for more than 6 hours, uncomplicated biliary colic is unlikely.

As inflammation in the gallbladder wall progresses, the poorly localized visceral pain gives way to moderately severe parietal pain that usually becomes localized to the right upper quadrant. Less commonly, the back may be the site of maximal pain (Boas sign); rarely the chest is the site.

Nausea with some vomiting is characteristic of acute cholecystitis, but these symptoms almost invariably follow rather than precede the onset of pain.

Fever is common (because of active inflammation in the gallbladder mucosa) but is usually less than 102°F unless gangrene or perforation of the gallbladder has occurred. Mild jaundice is present in 20% of patients and 40% of elderly patients. The jaundice is often subtle, and serum bilirubin concentrations usually are less than 4 mg/dL.Higher bilirubin levels suggest the

possibility of CBD stones, which may be found in one half of patients who have acute cholecystitis and jaundice. Another cause of pronounced jaundice in acute cholecystitis is Mirizzi's syndrome.

The abdominal examination often reveals right subcostal tenderness, with a palpable gallbladder in one third of patients. A palpable gallbladder is more common in patients who are having a first attack of acute cholecystitis, because repeated attacks usually result in a scarred fibrotic gallbladder that is unable to distend. For unclear reasons, the gallbladder is usually palpable lateral to its normal anatomic location.

A relatively specific finding for acute cholecystitis is Murphy's sign. During palpation in the right subcostal region, pain and inspiratory arrest may occur when the patient takes a deep breath that moves the inflamed gallbladder into contact with the examiner's hand. Left untreated, approximately 10% of cases are complicated by localized perforation and 1% by free perforation and peritonitis

DIAGNOSIS (Table (d))

Leukocytosis with a left shift is common. Because CBD stones with cholangitis are usually in the differential diagnosis, attention often is directed to liver biochemical testingThe serum bilirubin concentration also may be mildly

elevated in the range of 2 to 4 mg/dL, and even serum amylase and lipase levels may be elevated nonspecifically. A serum bilirubin level greater than 4 mg/dL or an amylase level greater than 1000 U/dL usually indicates coexisting CBD obstruction or acute pancreatitis and warrants further evaluation.

When the degree of leukocytosis exceeds 15,000 cells/mm^3, particularly in the setting of worsening pain, high fever (greater than 102°F), and chills, suppurative cholecystitis (empyema of the gallbladder) or perforation should be suspected, and urgent surgical intervention may be required.

Ultrasonography is the single most useful imaging study in acutely ill patients who have right upper quadrant pain and tenderness. Not only can it accurately establish the presence or absence of gallstones,

The positive predictive value of an ultrasonographic Murphy's sign is greater than 90% in detecting acute cholecystitis if gallstones are also present.

Ultrasonography can detect nonspecific findings of acute cholecystitis, such as pericholecystic fluid and thickening of the gallbladder wall to more than 4 mm. Both findings lose specificity for acute cholecystitis if ascites or hypoalbuminemia (less than 3.2 g/dL) is present.

The greatest utility of cholescintigraphy in these patients is to exclude acute cholecystitis and allow the clinician to focus on nonbiliary causes of acute abdominal pain.

With respect to acute cholecystitis, abdominal CT is most useful not in confirming the presence of acute cholecystitis but in detecting complications such as emphysematous cholecystitis or perforation of the gallbladder and in excluding other intra-abdominal abnormalities that may have a similar clinical picture. For example, abdominal CT is highly sensitive for detecting pneumoperitoneum, acute pancreatitis, pancreatic pseudocyst, hepatic or intra-abdominal abscesses, appendicitis, or obstruction or perforation of a hollow viscus. In a straightforward case of acute cholecystitis, an abdominal CT scan usually is not warranted; however, if the diagnosis is less certain or the optimal timing of surgery is in doubt, CT may be invaluable.

DIFFERENTIAL DIAGNOSIS[5,18,24]

The principal conditions to consider in the differential diagnosis are appendicitis, acute pancreatitis, pyelonephritis or renal stone, peptic ulcer disease, acute hepatitis, pneumonia, hepatic abscess or tumor, and gonococcal perihepatitis

Acute appendicitis is often confused. In general, fever, leukocytosis, and tenderness progress more inexorably in appendicitis. Complete abdominal ultrasonography can usually distinguish these two entities.

Acute pancreatitis also may be difficult to distinguish. Generally, vomiting is more prominent in acute pancreatitis, and hyperamylasemia is more profound.

Diseases of the right kidney may produce pain and tenderness similar to that of acute cholecystitis .Whereas the pain of uncomplicated peptic ulcer disease is usually chronic in nature and seldom confused with the pain of acute cholecystitis, a perforated ulcer may, at least initially, mimic severe acute cholecystitis. Signs of generalized peritonitis or a pneumoperitoneum strongly suggest a perforated viscus or at least the need for an emergency laparotomy.

Pneumonia with pleurisy may cause abdominal pain and tenderness, but the pleuritic nature of the pain and the chest radiographic result should be helpful in diagnoses.

In some instances, acute hepatitis, especially when caused by alcohol, may be accompanied by rather severe right upper quadrant pain and tenderness. Fever and leukocytosis add to diagnostic confusion with acute cholecystitis.

Gonococcal perihepatitis (Fitz-Hugh–Curtis syndrome) produces right upper quadrant pain, tenderness, and leukocytosis, which often overshadow any pelvic complaints.

Hepatic abscesses and tumors usually can be differentiated from acute cholecystitis on the basis of ultrasonographic findings

CHOLEDOCHOLITHIASIS[1,5,6]

Choledocholithiasis is defined as the occurrence of stones in the CBD. As do stones in the gallbladder, choledocholithiasis by itself may remain

asymptomatic for years, and the clinically silent passage of stones from the CBD into the duodenum is known to occur. Stones in the CBD, when they do cause symptoms, may lead to life-threatening complications

ETIOLOGY

Gallstones may pass from the gallbladder into the CBD or form de novo in the duct. Most pigment stones in the CBD are the softer, so-called brown pigment stones that form de novo in the CBD as a result of bacterial action on the phospholipid and bilirubin in bile. They often are found proximal to biliary strictures and frequently are associated with cholangitis.

Stones in the CBD usually come to rest at the lower end of the ampulla of Vater. Obstruction of the CBD increases bile pressure proximally and causes the ducts to dilate. Normal pressure in the duct is 10 to 15 cm H_2O and rises to 25 to 40 cm H_2O with complete obstruction. When pressure exceeds 15 cm H_2O, bile flow decreases, and at 30 cm H_2O it stops. .

CLINICAL MANIFESTATIONS

Acute obstruction usually causes biliary colic and jaundice, whereas obstruction that develops gradually over several months may present initially as pruritus or jaundice alone.

The physical examination is usually normal if obstruction of the CBD is intermittent. Mild to moderate jaundice may be seen when obstruction has been present for several days to a few weeks. Deep jaundice, particularly with a palpable gallbladder, suggests neoplastic obstruction of the CBD even when the patient has stones in the gallbladder. With long-standing obstruction, secondary biliary cirrhosis may result and lead to physical findings associated with chronic liver disease.

Laboratory studies may provide the only suggestion that choledocholithiasis is present. With bile duct obstruction, serum levels of both bilirubin and alkaline phosphatase increase. Bilirubin levels rise as a result of blocked excretion, whereas alkaline phosphatase levels rise as a result of increased synthesis of the enzyme by the canalicular epithelium.

Transient spikes in serum aminotransferase or amylase levels suggest passage of a CBD stone into the duodenum.

DIAGNOSIS[5,18]

Ultrasonography actually visualizes CBD stones in only approximately 50% of cases, whereas dilatation of the CBD to a diameter greater than 6 mm in is seen in approximately 75% of cases. Thus, ultrasonography suggest the presence of CBD stones but cannot definitively exclude them. EUS, although

clearly more invasive than standard ultrasonography, has the advantage of visualizing the CBD better with sensitivity and specificity rates of approximately 95%.

ERCP is the standard for the diagnosis of CBD stones, with sensitivity and specificity rates of approximately 95%. Percutaneous transhepatic cholangiography (PTC) is also an accurate means of confirming . PTC is most readily accomplished when the intrahepatic bile ducts are dilated and is now used primarily when ERCP is unavailable or unsuccessful.

Laparoscopic ultrasonography is a new imaging modality employed in the surgical suite immediately before mobilization of the gallbladder during cholecystectomy.

DIFFERENTIAL DIAGNOSIS

In patients who have jaundice, malignant obstruction or obstruction from a choledochal cyst may be clinically indistinguishable from choledocholithiasis.

Acute passive congestion of the liver, associated with cardiac decompensation, may cause intense right upper quadrant pain, tenderness, and even jaundice with serum bilirubin levels as high as 10 mg/dL or more.

Acute viral hepatitis rarely may cause severe right upper quadrant pain with tenderness and fever..

Acquired immunodeficiency syndrome (AIDS) cholangiopathy and papillary stenosis must be considered in patients infected with human immunodeficiency

CHOLANGITIS (BACTERIAL CHOLANGITIS)[4]

Of all the complications of gallstones, cholangitis kills most swiftly. Pus under pressure in the bile ducts leads to rapid spread of bacteria, via the liver, into the blood, and resulting septicemia.

Cholangitis is relatively common in patients with choledocholithiasis, nearly universal in patients who have post-traumatic bile duct stricture.

The bacterial species most commonly cultured are *Escherichia coli,* enterococci, and *Klebsiella, Pseudomonas,* and *Proteus* species. Anaerobic species such as *Bacteroides fragilis* or *Clostridium perfringens* are found in about 15% of appropriately cultured bile specimens. Anaerobes usually accompany aerobes, especially *E. coli*.

CLINICAL MANIFESTATIONS

Classic Charcot's triad of pain, jaundice, and fever is the hallmark of cholangitis. . Mental confusion, lethargy, and delirium may be the only features of the history obtainable, particularly in elderly patients.

Physical examination fever is nearly universal, occurring in 95% of cases. Right upper quadrant tenderness occurs in approximately 90% of

patients, whereas jaundice is clinically detectable in only 80%. Peritoneal signs are found in only 15%. In severe cases, hypotension and mental confusion may coexist. In most cases, blood culture results are positive for enteric organisms, especially if cultures are obtained during chills and fever spikes

DIAGNOSIS

The principles of radiological diagnosis are the same as those for choledocholithiasis.

INVESTIGATIONS FOR CHOLECYSTITIS

Consists of imaging and laboratory investigations (Table (c))

IMAGING STUDIES OF THE BILIARY TRACT [2,5,18,24]

With the possible exception of ultrasonography, none of the tests should be ordered routinely in the evaluation of a patient with suspected cholecystitis. Rather, the diagnostic evaluation should proceed in a rational stepwise fashion based on the individual patient's symptoms, signs, and laboratory results.

Plain abdominal films lack both sensitivity and specificity. Plain abdominal films have their greatest utility in evaluating patients who have unusual complications of gallstones, such as emphysematous cholecystitis, cholecystoenteric fistula, and a porcelain gallbladder.

ULTRASONOGRAPHY (Plate 1a, b)

It requires no special preparation of the patient, involves no ionizing radiation, is simple to perform, and provides accurate anatomic information.

Ultrasonography of the gallbladder should follow a fast of at least 8 hours, The diagnosis relies echogenic objects within the lumen of the gallbladder that produce an acoustic shadow. Modern ultrasonographic equipment can routinely detect stones as small as 2 mm in diameter.

The overall sensitivity of ultrasonography for the detection of gallstones in the gallbladder is greater than 95% for stones larger than 2 mm in diameter.The specificity is greater than 95% when stones are seen with an accompanying acoustic shadow. The contracted gallbladder filled with stones may give a "double-arc-shadow" or "wall-echo-shadow" sign, with the gallbladder wall, echogenic stones, and acoustic shadowing seen in immediate proximity Ultrasonography is the gold standard for the diagnosis of gall stones Finally, ultrasonography has substantial utility in the diagnosis of acute cholecystitis. Pericholecystic fluid, when seen in the absence of ascites, and thickening of the gallbladder wall to greater than 4 mm (in the absence of hypoalbuminemia) are nonspecific findings that are suggestive of acute cholecystitis. A more specific finding is the so-called ultrasonographic Murphy's sign, in which the ultrasonographer elicits focal gallbladder tenderness under the transducer

ENDOSCOPIC ULTRASONOGRAPHY

Has the advantage of being able to visualize the CBD and thus confirm or exclude the presence of choledocholithiasis with a high degree of accuracy

ORAL CHOLECYSTOGRAPHY

Limited application as a secondary means of identifying stones in the gallbladder

OPERATIVE CHOLANGIO GRAPHY

INDICATIONS **:**

1. **Gall stone disease and post cholecystectomy cases**
2. **Laparotomy for jaundice –mechanical obstruction suspected.**
3. **Traumatic biliary stricture and post traumatic bilary stricture**

CHOLESCINTIGRAPHY (HEPATOBILIARY SCINTIGRAPHY)

(Plate 3 b)

Cholescintigraphy is a radionuclide based imaging test of the gallbladder and biliary tract that has its greatest utility in the evaluation of patients suspected of having acute cholecystitis. The test can be performed on an emergency basis in a nonfasting patient after the intravenous administration of a gamma emitting Tc-labeled iminodiacetic acid derivative (e.g., hydroxy iminodiacetic acid [HIDA], diisopropyl iminodiacetic acid [DISIDA]) that is rapidly taken up by the liver and excreted into the bile.

ENDOSCOPIC RETROGRADE CHOLANGIO PANCREATICOGRAPHY

Just as ultrasonography is the gold standard for the diagnosis of cholelithiasis, ERCP has now become the gold standard for the diagnosis of choledocholithiasis. Stones appear as filling. The overall specificity of ERCP for the detecting CBD stones is approximately 95%. **(Plate 3b)**

Its therapeutic applications have revolutionized the treatment of patients with choledocholithiasis

COMPUTED TOMOGRAPHIC CHOLANGIOGRAPHY AND MAGNETIC RESONANCE CHOLANGIOGRAPHY

Both computed tomographic cholangiography (CTC) and magnetic resonance cholangiography (MRC) are powerful imaging techniques for evaluating patients with intra-abdominal abnormalities.

The correlation between the findings of MRC and ERCP was greater than 90%. CTC and MRC have the advantage of being noninvasive but obviously offer no therapeutic potential. Therefore, they may be most useful in excluding choledocholithiasis (either preoperatively or postoperatively) in patients thought to have a low probability of having stones in the CBD, and ERCP may be reserved for those with a higher probability of having CBD stones.

ACUTE ACALCULOUS CHOLECYSTITIS[1,2,18]

DEFINITION

Acute acalculous cholecystitis is acute inflammation of the gallbladder in the absence of stones. The term *necrotizing cholecystitis* has been proposed to reflect the distinct etiology, pathology, and prognosis of the disease

EPIDEMIOLOGY

Acute acalculous cholecystitis accounts for 5% to 10% of cholecystectomies.

CONDITIONS ASSOCIATED COMMONLY:

1 Sepsis.

2. Hypotension

3. Burns

4. Major trauma

5. Gasteroentritis

6. TPN

7. Kawasaki disease and other autoimmune vasculitis

8. Typhoid fever(common in tropics)

9. Viral fever eg. Dengue

PATHOGENESIS

Most cases of acute acalculous cholecystitis occur in the setting of prolonged fasting, immobility, and hemodynamic instability. The gallbladder epithelium, although normally a robust tissue, is exposed to one of the most

noxious environments in the body: a concentrated solution of bile acid detergents designed to solubilize lipids.

Thus, in an immobile, fasting patient with splanchnic vasoconstriction resulting from septic shock (often, a patient in the intensive care unit), the stage is set for an ischemic and chemical injury to the gallbladder epithelium.

CLINICAL MANIFESTATIONS

The clinical features of acute acalculous cholecystitis differ from those of acute cholecystitis caused by stone disease. Although right upper quadrant pain, fever, localized tenderness overlying the gallbladder, and leukocytosis may be evident in classic presentations

By the time the diagnosis has been made, at least one half of the patients have experienced a complication of cholecystitis, such as gangrene or a confined perforation of the gallbladder. Empyema and ascending cholangitis may further complicate cases in which bacterial superinfection of the gallbladder has occurred.

DIAGNOSIS

The rapid development of complications in acute acalculous cholecystitis makes early diagnosis critical for avoiding excessive mortality.

ULTRASONOGRAPHY (Plate 1b)

Three ultrasonographic findings that point to gallbladder disease include a thickened gallbladder wall (defined as >4 mm) in the absence of ascites or hypoalbuminemia, a sonographic Murphy's sign (defined as maximum tenderness over the sonographically localized gallbladder), and a pericholecystic fluid collection.

COMPUTED TOMOGRAPHY (CT)

CT findings suggestive of cholecystitis include wall thickening (>4 mm), pericholecystic fluid, subserosal edema (in the absence of ascites), intramural gas, and sloughed mucosa. The sensitivity and specificity of these findings for predicting acute acalculous cholecystitis at surgery have been reported to exceed 95%.

HEPATOBILIARY SCINTIGRAPHY

Hepatobiliary scintigraphy has proven useful in excluding cystic duct obstruction in patients with other clinical features suggestive of acute cholecystitis. Under normal conditions, the radionuclide is taken up by the liver, secreted into bile, concentrated in the gallbladder (where it produces a "hot spot"), and emptied into the duodenum. A positive scan for cystic duct obstruction is defined as failure to fill the gallbladder despite the normal passage of radionuclide into the duodenum

In light of rapid progression of acute acalculous cholecystitis to gangrene and perforation, early recognition and intervention are required. Supportive medical care should include restoration of hemodynamic stability and antibiotic coverage for gram-negative enteric organisms and anaerobes if biliary tract infection is suspected.

TREATMENT OF CHOLECYSTITS[1,8,11,12]

Treatment options available are

1. **Surgical (a) open (b) laparoscopic**
2. **Conservative followed by elective cholecystectomy after 6 weeks**

Before a decision can be made regarding the appropriate therapeutic approach, the stage of disease must be defined.

There are three stages of cholelithiasis:

(1) The asymptomatic stage,

(2) The symptomatic stage without complications, and

(3) The symptomatic stage with complications, such as acute cholecystitis, choledocholithiasis, biliary pancreatitis, gallbladder cancer, and gallstone ileus

PRINCIPLES OF NON-OPERATIVE TREATMENT[4]

1. **Naso gastric aspiration and IVF administration.**
2. **Administration of analgesics.**
3. **Administration of antibiotics**

It consists of :

1. **Rest to gall bladder by nil oral, IV fluids , and continuous Ryles tube aspiration.**
2. **Pain is relieved by I.M. Pethidine with atropine IV or propantheline to knock off vagal influence.**
3. **Broad spectrum antibiotic effective against Gram –ve and anaerobic organisms are given (eg. Cefzolin, Cefuroxime with Gentamycin and Metranidizole) .**
4. **During the first 48 hours monitoring of vitals are important, along with investigations to find out the cause and other systemic diseases.**

INDICATIONS FOR STOPPING NON OPERATIVE TREATMENT

1. **Failure to improve after 48 hours of therapy.**
2. **Tender, enlarging mass in right hypochondrium.**
3. **Development of rigors**
4. **Features of generalized peritonitis**
5. **Uncertainty about diagnosis.**

ADVANTAGE OF EARLY SURGERY DONE IN 48-72 HOURS OF UNCOMPLICATED ACUTE CHOLECYCTITIS IS

1. **Short hospital stay.**

2. **Prevention of development of serious complications eg. Empyema.**
3. **Associated with fewer technical problems and mortality**
4. **Prevention of future recurrence.**

SURGICAL TREATMENT

Patients with complicated gallstone disease, including acute cholecystitis, gallstone pancreatitis, and choledocholithiasis, were more likely to require an open procedure or conversion from a laparoscopic to an open approach than were patients with uncomplicated disease

OPEN CHOLECYSTECTOMY [11, 12]

TECHNIQUE

With the surgeon standing on the patient's right side,

Kocher's incision is made (2 finger breadths below right costal margin starting from lateral border of rectus sheath) or a midline incision can be made

Exploration of abdomen done

Adhesions to gall bladder were removed carefully

After opening the peritoneum over Calot's triangle cystic artery and duct were identified and ligated separately

Then gall bladder can be removed by 2 approaches

1. **Fundus first**
2. **Duct first approach**

Gall bladder removed from gall bladder fossa of liver after dividing the peritoneal reflections the and homeostasis achieved

Drainage tube is not routinely kept

LAPAROSCOPIC CHOLECYSTECTOMY[8, 25, 28]

The driving force initially was patient demand. With the minimally invasive approach, minimal scarring, reduced pain, and quicker return normal activities were recognized.

TECHNIQUE

Preparation of the patient done. Prophylactic anitibiotics are not routinely administered. Patients with potenital infectious complications of gall stones, including acute cholecystitis and cholangitis, should receive antibiotics. Pneumoperitoneun created with a nonflammable gas such as carbon dioxide. A trocar is placed at the umbilicus and a telescope is introduced. Three additional trocars are placed.. The assistant retracts the gall bladder fundus cephalic side, and the cystic duct and artery identified separately. Special care must be taken to identify the junction of the cystic duct and gall bladder. If the anatomy is clear and no evidence of choledocholithiasis is seen, the cystic duct and cystic artery are divided between metal clips. The gallbladder is then dissected from the liver bed and delivered through the umbilical incision. Care is taken to avoid perforation and spillage of gallstones.

EMERGENCY LIFE SAVING PROCEDURES THAT ARE DONE ARE

- **Cholecystostomy.**
- **Percutaneous cholecystostomy**

COMPLICATIONS:(5%)

Per-operative:

1. **Bleeding**
2. **Bile duct injury**
3. **Perforation of transverse colon**
4. **Bile stone spillage into peritoneal cavity.**

Post-operative :

- Bile leakage
- Periumblical haematoma
- Right basal lung collapse
- Subphrenic abscess.
- Fever
- Sepsis
- Peritoneal collection
- Pancreatitis/ biliary stricture

NON OPERATIVE TREATMENT[4, 13, 2]

This treatment consists of

GALL STONE DISSOULTION THERAPY

A. ORAL DISSOLUTION THERAPY :

Indications

Functioning gall bladder

Radiolucent stones

Stone less than 2 cm in diameter

Patient unfit for surgery

Method : Chenodeoxycholic acid or urodeoxycholic acid orally . In stones <5mm it took 6 months of treatment , due to side effects like diarrhea , it is avoided presently**.**

B. ALTERNATE TREATMENTS THAT HAVE BEEN TRIED ARE

1. Citrate 2.Monotrepenes etc. but all have failed to give successful results

C. EXTRACORPORAL SHOCK WAVE LITHOTRIPSY

ESWL has been tried by Sackmann *et al* but the complications like skin petechiae, biliary colic , abdominal wall tenderness, nausea, cardiac arrthymias, hamaturia and acute cholecystitis were reported . This form of treatment is not widely practiced nowadays.

3. MATERIALS AND METHODS

Study design : Descriptive study

Place of the study : Kilpauk Medical College Hospital

Period of the study ; March 2004- February 2006

Sample specifications :

Inclusion criteria

i. 34 consecutive patients (male & female) admitted in KMCH with the diagnosis of Acute Cholecystitis by clinical and USG

Exclusion criteria

i. Other causes of acute abdominal pain including acute appendicitis, peptic ulcer disease, renal colic,acute pancreatitis,intestinal perforation, basal pneumonitis, perihepatitis
ii. Biliary pain lasting less than 6 hours (biliary colic)
iii. S.amylase > 5 fold rise or 1000 U/ dl
iv. S.Bilirubin > 10 mg/dl

Evaluation of the case

34 adult patients admitted in the surgical ward of KMCH with the diagnosis of acute cholecystitis were included in the study.

Detailed history including name, age, sex, nativity, presenting symptoms, dietary habits, family history, drug history, previous abdominal surgery, relevant medical conditions were recorded.

Description of symptoms with chronology, associated features like fever, chills, vomiting, scleral icterus, high coloured urine, past medical history were made meticulously.

Clinical examination including sensorium, vitals, icterus, pallor, temperature, rigors, were sought after and recorded. Systemic examination including abdomen, cardiovascular , respiratory and neurological systems were done.

The following investigations were done in the same order.

i. X ray chest PA, abdomen erect

ii. ECG
iii. CBC
iv. Urea, creatinine, sugar
v. Sr. electrolytes
vi. USG abdomen
vii. Sr.amylase
viii. Liver function test
ix. Upper GI endoscopy
x. Blood culture

Following investigations were done whenever required

i. CT scan abdomen
ii. ERCP
iii. Stone analysis
iv. Bile culture

The patients were followed up. Those patients who were managed conservatively were observed. For those who underwent surgery, details about the type of surgery, per operative findings, complications of surgery, and short term follow up of 1 week were recorded.

5. OBSERVATIONS

EPIDEMIOLOGY [14, 20, 27]

Of the studied 34 patients, females predominated (70%). Half (5) of the male patients had co-morbid / predisposing factors like diabetes mellitus, obesity, haemolytic anaemia, chronic liver disease etc. On contrary 67% females had predisposing factors. Most of the cases in the female group were in the age group less than 50 years (Table 1 and Figure 2.1a).

Table 1:

SEX	Age < 50	AGE > 50	COMORBID FACTORS
MALE	5	5	5
FEMALE	18	6	12

ETHNIC DISTRIBUTION [4, 20, 27]

Our study group comprised of predominantly Tamilian population (25) though few patients belonged to Telugus (2) , Punjabis(1), Sourashtrians(1), Nepalis (1). Mixed stones accounted for 68% of Tamil population whereas cholesterol stones predominated in North Indian patients (Table 2 and Figure 2.1b).

Table 2:

ETHNIC GROUP	NUMBER OF PATIENTS	PIGMENT STONES	CHOLESTEROL STONES	MIXED STONES
Tamilians	25	5	6	14
Telugus	2	0	0	2
Punjabis	1	0	1	0
Sourashtrians	1	0	1	0
Nepalis	1	0	0	1

CO-MORBID CONDITIONS[6, 17]

Out of the 34 patients studied 41% of the patients were obese, and more than 23% had diabetes mellitus and , 6% of the patients had haemolytic anaemia who later found to have black pigment stones. 2 of the patients were pregnant and they were managed conservatively. 12% of the patients who developed calculous cholecystitis were on oral contraceptive pills containing

estrogen and 1 of them was on lipid lowering drug. The details were given in the pie chart (Fig. 2.3b and Plate 8a).

CLINICAL PRESENTATION [1, 25]

Abdominal pain was the universal presentation (100%), which was the defining criterion for the diagnosis. Loss of appetite and vomiting were the other common symptoms. Fever was seen in 76% of the cases, and jaundice in 53% of the patients Hypotension and shock were seen in 5 cases there was no mortality among study group (Table 3 and Figure 2.2a).

Table 3:

SYMPTOMS	NUMBER OF PATIENTS
ABDOMINAL PAIN	34 (100%)
LOSS OF APPETITE	30 (88.2%)
VOMITING	28 (82.3%)
FEVER	26 (76.4%)
JAUNDICE	18 (52.9%)
SHOCK	5 (14.7%)

DIAGNOSTIC METHODS

IMAGING STUDIES:

Abdominal and chest X-rays though not diagnostic themselves were helpful in excluding other causes like hollow viscus perforation and basal pneumonia.

ULTRASONOGRAM [13, 18, 24]

Ultrasonogram was diagnostic in all cases(100%). – Findings observed are Gall stone with /without sludge in 30 cases(88%) (calculus cholecystitis).Gall bladder wall thickening observed in 28 cases(82%). Pericholecystitic fluid collection in 27 cases(79%). Sonographic Murphy's sign in 32 cases IHBR dialatation indicative of coexistent choledocholithiasis in 3 cases, later verified by CT scan and ERCP.The findings of gall bladder stone and sonographic Murphy's sign were the most sensitive USG observations (Table 4; Plate 1 & 2).

Table 4:

SEX	GB STONES/ SLUDGE	WALL THICKNESS > 4 MM	PERICHOLE-CYSTIC FLUID COLLECTION	IHBR &CBD DILATATION	SONOGRAPHIC MURPHY'S SIGN
MALE	7	8	7	1	10
FEMALE	23	20	20	3	22

CT SCAN AND ERCP: [4, 18, 24]

CT scan was done in 10 cases either to confirm diagnosis or to exclude closely mimicking conditions like acute pancreatitis common bile duct stones and small perforations. CT picked up acute cholecystitis in all cases done (100%). No false positives were observed with USG when compared with CT.

ERCP was done in 3 patients - pre operative in 1 patient and post operative in 2 patients (suspected CBD stone –1 and bile leak –1). All patients who had CBD stone by CT scan were proved to have the same by ERCP (100%).

ERCP was able to detect the site of bile leak in 1 patient subjected for the same (Table 5 and Plate 3) .

Table 5:

SEX	PRE OPERATIVE ERCP	POST OPERATIVE ERCP	USG - CBD STONE	CT – CBD STONE
MALE	1	1	1	1
FEMALE	2	2	1	2

LIVER FUNCTION TESTS[5, 13, 24]

Liver functions tests are performed in all cases. The results are given as pie diagram (Figure 2.3).

- **Serum AST and ALT are elevated less than 3 folds in most of the cases, patients who had cholidocholithiasis had higher levels transferases.**
- **Serum bilirubin was raised in 82.3 % of the cases. In 4 patients the raise was more than 3 mgm which later proved to be because of cholidocholithiasis**

- **Serum amylase level was found normal in only 5 cases(14.7) . In rest of them the raise was less than 5 folds . Patients who had more than 5 fold raise of Serum amylase (biliary pancreatitis) were excluded from the study.**

TREATMENT [1, 8, 15, 28]

30 patients (10: 20) were surgically treated after initial stabilization with fluids, antibiotics, nil oral. Surgery was done within 1 week of symptoms in all patients (mean 6 days). 20 cases(58.8%) were done by open method and 10 cases by laproscopic method.4 patients were treated on conservative lines either because of rapid resolution of symptoms or associated severe co morbid conditions (DM, IHD, Pregnancy). 1 pregnant mother (first trimester) was treated conservatively (Table 6 and Figure 2.2b and Plate 7).

Table 6:

SEX	SURGERY		CONSERVATIVE*
	OPEN	LAPROSCOPY	
MALE	6	4	0
FEMALE	14	6	4

*Surgical treatment offered after an interval of > 6 weeks

SURGICAL FINDINGS [8, 11, 12, 16]

Out of the 30 cases taken for surgery 93.3% of the cases had stones in the

gall bladder, and 3 patients(10%) there was no calculous found . There was dense adhesions present in 13% of the patients ,one of the patients had a perforated gall bladder with pus in peritoneal cavity (Table 7; Figure 2.5 and Plate 4, 5, 6 and 9).

Table 7:

FINDINGS	**NO. OF CASES(%)**
CALCULI	28 (93.3%)
ACALCULUS CHOL.	3 (10%)
EMPHYSEMATOUS GB	1(3.3%)
DENSE ADHESIONS	4(13.3%)
PERFORATION	1(3.3%)
GANGRENE	1 (3.3%)

POST OPERATIVE FOLLOW UP[4, 29]

On follow up for 1 week period, the following were observed. Most of the patients who underwent surgery by open or laproscopic method had good recovery. Fever was the most common post-operative complication in both forms of surgery. Bile duct injury observed in 5% (1) of cases of open cholecystectomy but none were reported in laparoscopic cholecystectomy. Wound infection occurred at the rate of 30 % in open cholecystectomies. Since

the patients were not followed up for more than 1 week long term complications post cholecystectomy were not dealt in this study (Table 8).

Table 8:

COMPLICATIONS	OPEN CHOLECYSTECTOMY	LAP CHOLECYSTECTOMY
UNEVENTFUL	10	6
BILE DUCT INJURY	1	0
FLUID COLLECTION	1	0
FEVER	8	4
SEPSIS	2	1
WOUND INFECTION	6	0

DISTRIBUTION OF STONES [10, 21]

Of the 30 stones studied 16% was pigment stones and 50%the stones were found to be cholesterol stones and the remaining were mixed stones, all three varieties of stones were found more in females (Table 9 and Figure 2.3a and Plate 8a).

Table 9:

	PIGMENT	CHOLESTEROL	MIXED
MALE	2	2	3
FEMALE	2	12	7

ORGANISMS GROWN IN BILE CULTURE[5, 8, 10]

Bile was sent for culture in 15 cases.

The results showed no growth in 53% of cases and E. coli was the commonest organism identified. More than 50 % of cases there was no growth in both aerobic and anaerobic culture.

Table 11: **ORGANISMS GROWN IN BILE CULTURE**

ORGANISMS	N= 15	%
Sterile	8	53.3
E. coli	4	26.6
Klebsiella sp	1	6.6
Enterobacter	1	6.6
Salmonella sp	1	6.6
Anaerobes	0	0

6. DISCUSSION

We compared our epidemiological data with one of the largest multicentric Indian series by V.Jayanthi , Prasanthi R, Surendran R Palanivelu C et al published in BHJ[35].

ETIOPATHOGENESIS

Their study concluded that women tend to outnumber males and are younger than their counterparts, which is also observed in our study. Our study comprised of predominant Tamilian population. Mixed stoned accounted for 68% of South Indian cases where cholesterol stones predominated in North Indian cases. Jayanthi. V observed that 80% were pigment stones and 6.9% mixed stones in south Indian population.

Trotman et al[37] observed no difference between blacks and whites in stone composition. Kanfman et al showed black predominance of pigment stones North America. Diehl et al[36] also had similar conclusions. A change in dietary pattern and life style has a role in distribution of stone. GREPCO[34] study in Italy came out with similar conclusions.

CO-MORBID CONDITIONS

According to Jayanthi V et al parity, alcohol, smoking had no effect on the incidence of gallstones in women. They observed a slight protective effect of smoking in males. Other studies by Sarin SK [32]. claimed higher incidence

in multiparous women and in positive family history. Diehl et al[36] observed no such effect. Diabetes as a contributing factor was not confirmed convincingly in all these studies. Obesity was a major associated factor in more than 70% of the cases of calculus cholecystitis in study by Palanivelu C[35] et al., which was comparable with our study group which had obesity in 74% of cases of calculus cholecystitis.

Our series had 23% incidence of diabetes mellitus. The risk factor for cholecystitis in diabetics is probably more pronounced in females than males. Multivariate analysis showed it to be statistically significant ($p = 0.02$).

Acalculus cholecystitis contributed to about 7-22% in a different series as complied by David Lane J[38]. Majority of the cases occurred in postoperative set up and critically ill patients. In our study the incidence was in 4 (13.3%) of cases, of which 2 of the cases occurred in postoperative setting and 1 in critical care setting. The other patient had diabetes mellitus. The emphasis here is the high index of clinical suspicion and need for expedite evaluation in suspected cases.

DIAGNOSIS

With due respect to the diagnosis of acute cholecystitis this study validated the utility of ultra sonogram as the primary modality of choice. GB wall thickness, pericholecystitic fluid collection, sonographic Murphy's sign were sensitive and specific.

On comparing ultrasonogram and CT in our study, both were found to be sensitive and specific and correlated well (100%) of the patients. There were no false negatives or false positives.

On contrary an article in RSNA by Robert T. Harvey et al[39] observed that CT has sensitivity lesser than USG and USG picked up additional finding in 4% of cases missed by CT and led to change in management in 2.5% of cases. He observed that ultrasonogram rather than CT as the best diagnostic modality and CT is useful in difficult situations where differential diagnoses were confusing.

ERCP had good correlation with CT in our study closely, which was validated by other studies.

TREATMENT

In our series, operative treatment was offered to 30 (88.2%) patients immediately after diagnosis, and 4 (11.8%) patients were put on conservative line of management because of there general condition did not permit surgery. They were taken for surgery electively after a period of 6 weeks.

Our study was compared with the study by Jarvinen et al.[15] on early cholecystectomy for acute cholecystitis published in annals of surgery 1982 .

It showed good results were obtained for both open and laparoscopic cholecystectomy if surgery was carried out at the earliest after diagnosis is achieved (86%). If surgery was delayed after diagnosis complications were more(18%). Lesser complications were observed if the patients are taken for elective surgery after the acute episode (2%).

Open cholecystectomy was preferred over laproscopic cholecystectomy in acute setting in a study by Edlund Y,et al [25]1994 BMJ. Lujan Ja, et al[28] 1995 in Journal of American College of Surgery also preferred open cholecystectomy over laparoscopic cholecystectomy in acute cholecystitis, which was comparable with our study in which more than 66% of the cases were managed by open cholecystectomy.

Due to technical reasons, bile obtained from 15 patients only were subjected to culture, the reported major organisms grown in culture are aerobic gram negatives (E.coli- 26.6%). We did not document any anaerobic organisms, and 50% of the bile sent for culture was found to be sterile.

On the contrary the study by Ballal M, Jyoti KM, *et. al*[40]. published in the Indian Journal of Medical Microbiology reported a high incidence of

anaerobic organisms. This might be possibly explained by the difference in sample collection methods, use of pre operative antibiotics, and incubation policies.

POST-OPERATIVE FOLLOWUP

In our study the cases were followed up for a period of 1-week post operatively. Al-Haijar N, Duca S.,*et al*[41], did a similar study, where they followed up 1453 patients after cholecystectomy. We compared both the studies.

In our study 66% of patients had an uneventful recovery. Minor complications like fever and wound sepsis accounted for majority of post op complications. Bile duct injury was observed in 5% of the cases. Al- Haijar reported hemorrhage as the major complication (9.5%), followed by bile duct injury (6.9%). Mild postoperative symptoms occurred in 60 % (grade I as per Clavien's classification). The discrepancy in the results might be because of differences in the timing of surgery, local expertise in open cholecystectomies and post operative care between two studies.

7. CONCLUSIONS

- Women had higher incidence than men.
- In younger age group women are affected more than men, but in older age group both were comparable.
- Gall stones was the most common etiology observed. Mixed stones contributed to more than 50% of cases of acute cholecystitis in our series.
- Women who were affected have relatively increased prevalence of risk factors like obesity and diabetes mellitus.
- Abdominal pain was the universal presentation in all cases of acute cholecystitis.
- The course and the complications of acute cholecystitis in women with significant risk factors were comparable to men.
- Acalculous cholecystitis contributed to 13.3% of total cases and all the cases were either in postoperative period or critically ill patients.
- Ultrasonogram remains the most important investigation of choice for the diagnosis of acute cholecystitis. Computed tomography had good correlation with ultrasonogram. Computed tomography detected complications and common bile duct stones better than ultrasonogram.

- Liver function tests were not altered in majority of the cases with acute cholecystitis unless it is complicated by common bile duct stones.
- Open cholecystectomy was the most preferred and the commonest treatment option adopted in the patients in our study. Laparoscopic cholecystectomy was done in few patients. The complication rate was lesser in laparoscopic cholecystectomy than in open cholecystectomy.
- In more than 50 % of the cases the bile culture was sterile and in the remaining cases *Escherichia coli* is the most commonly isolated organism from bile culture in acute cholecystitis.
- And on short term follow up; most of the patients had an uneventful recovery. Although major complications like bile duct injury occurred in less than 5% of the patients, minor complications like fever and wound sepsis occurred in significant proportions.

Figure 2.1a

EPIDEMOLOGY

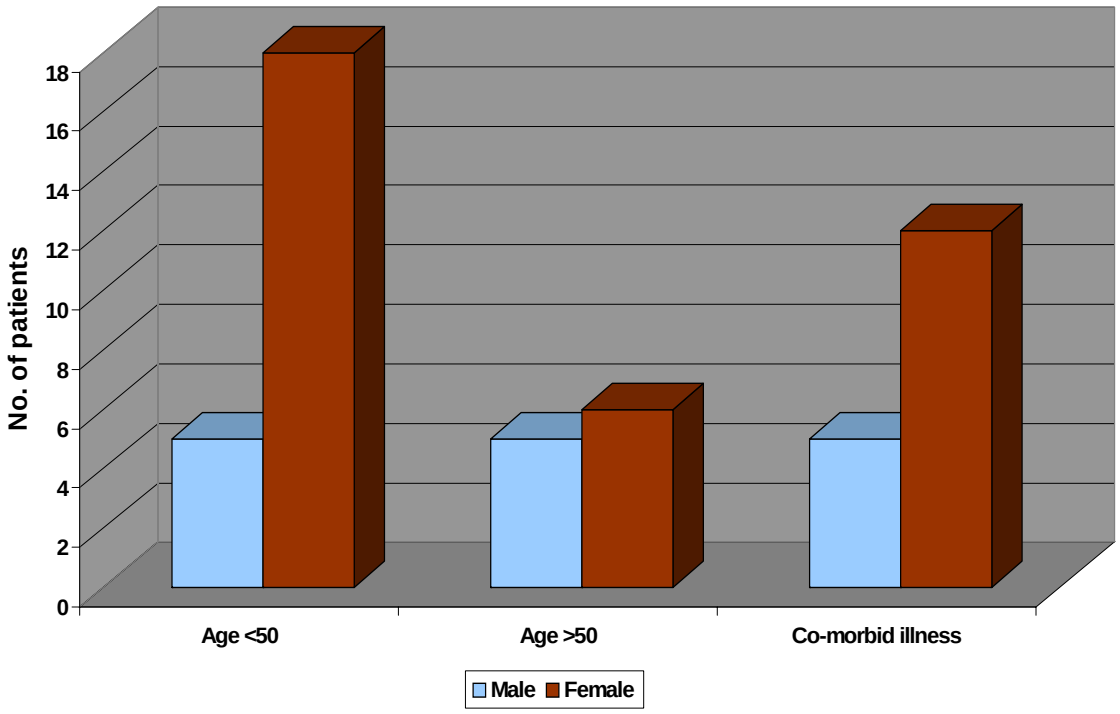


Figure 2.1b

ETHINIC DISTRIBUTION OF STONES

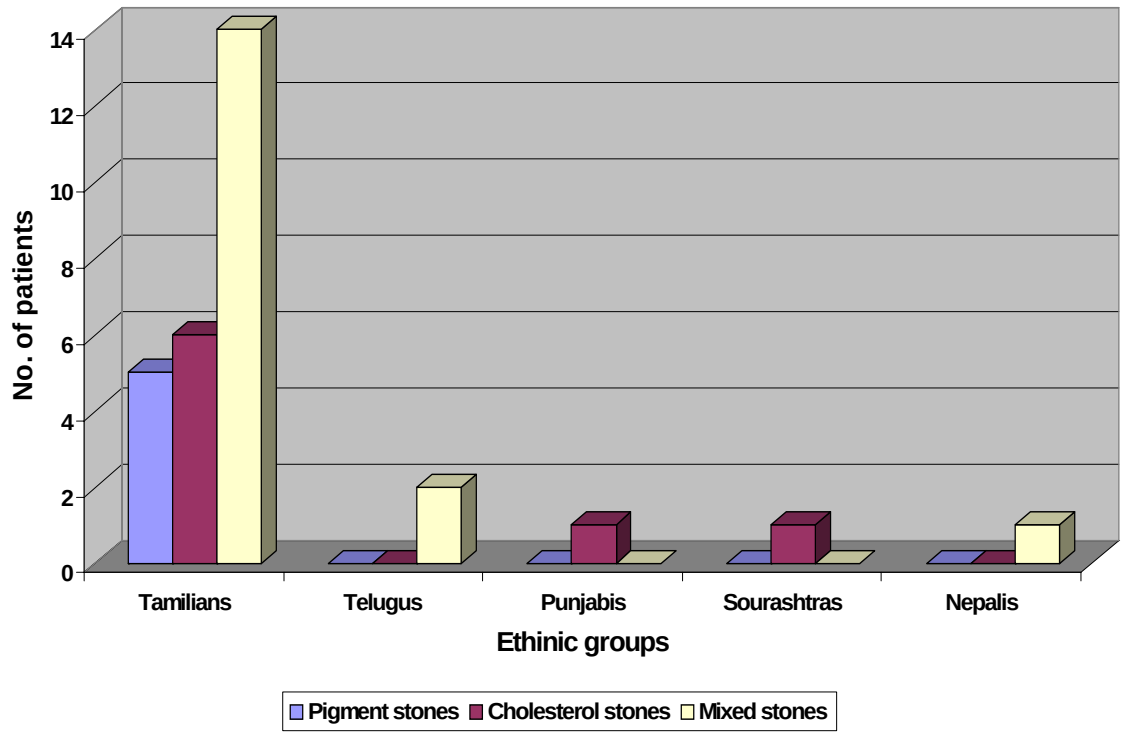


Figure 2.2a
CLINICAL PRESENTATION

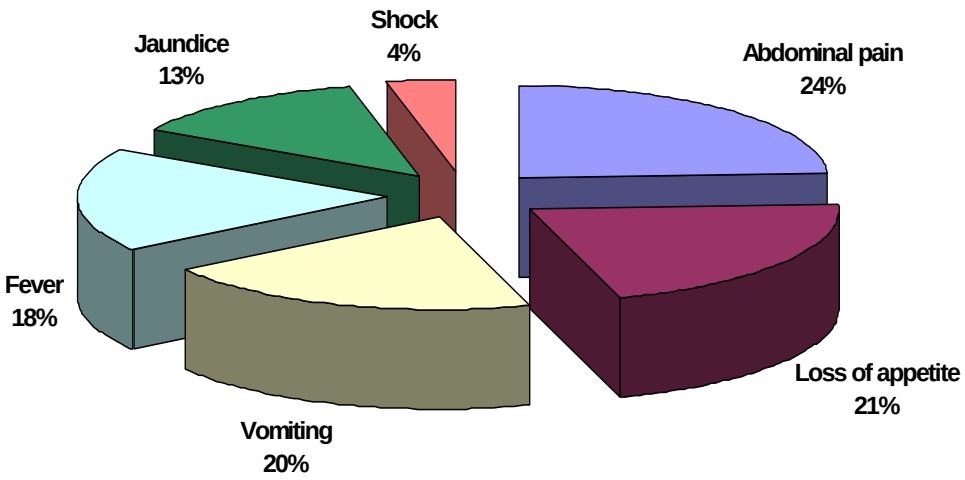


Figure 2.2b
TREATMENT OFFERED

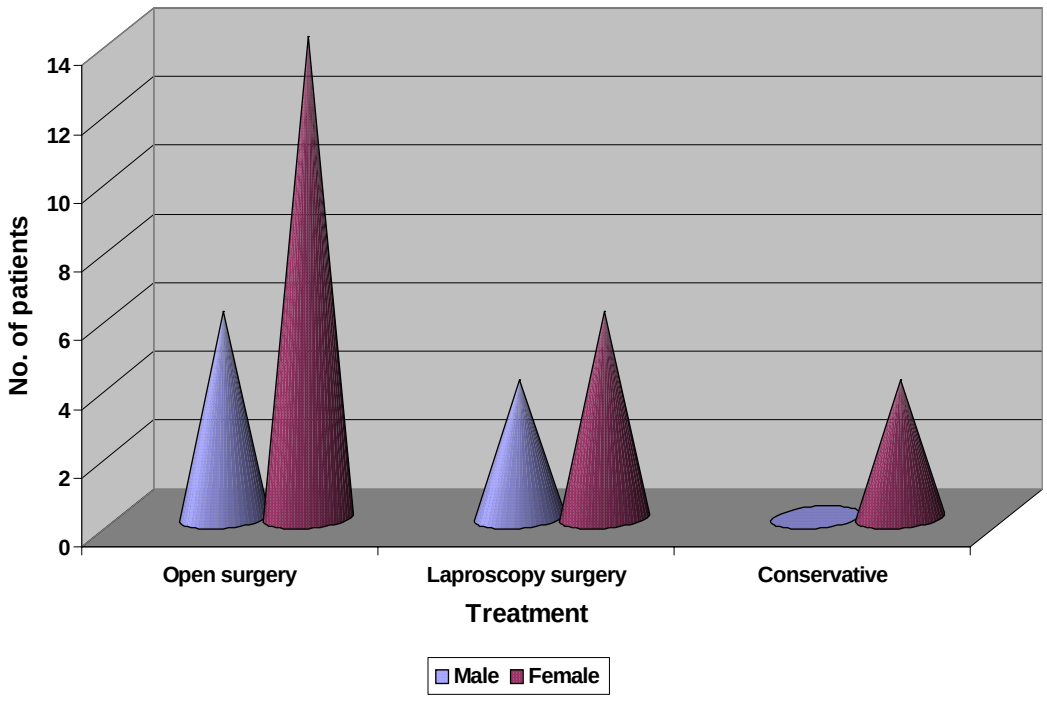


Figure 2.3a
STONE DISTRIBUTION

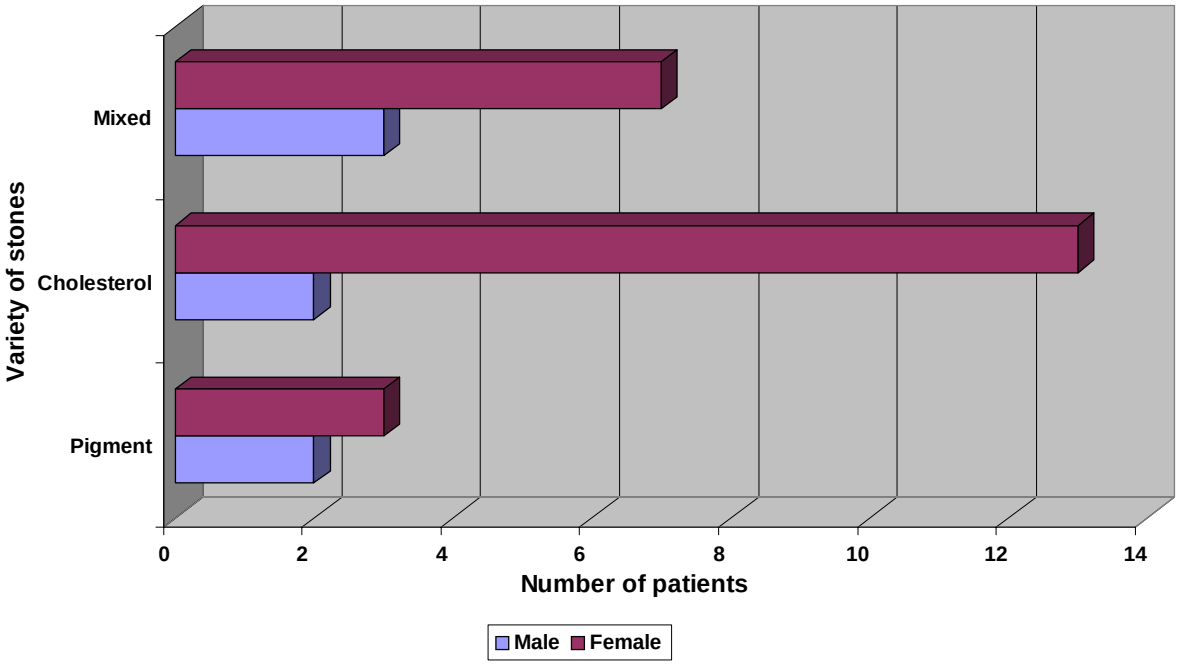


Figure 2.3b
CO-MORBID CONDITIONS

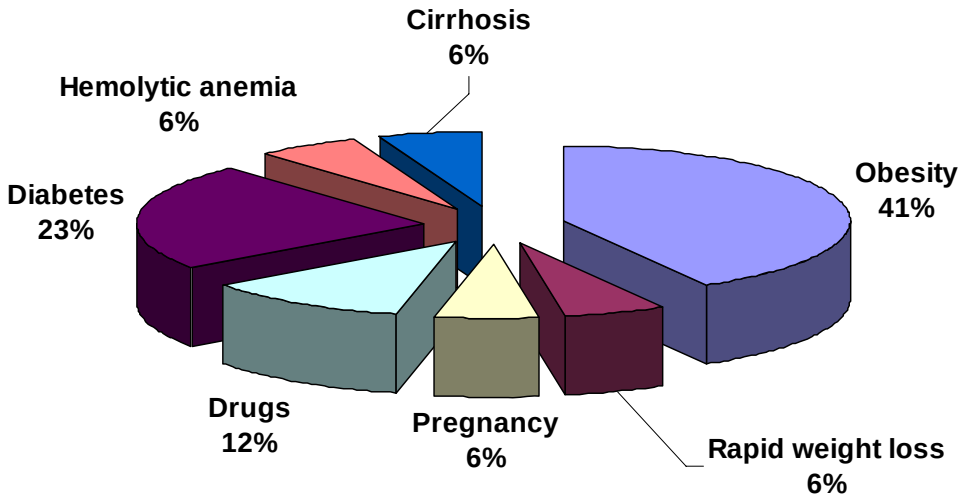
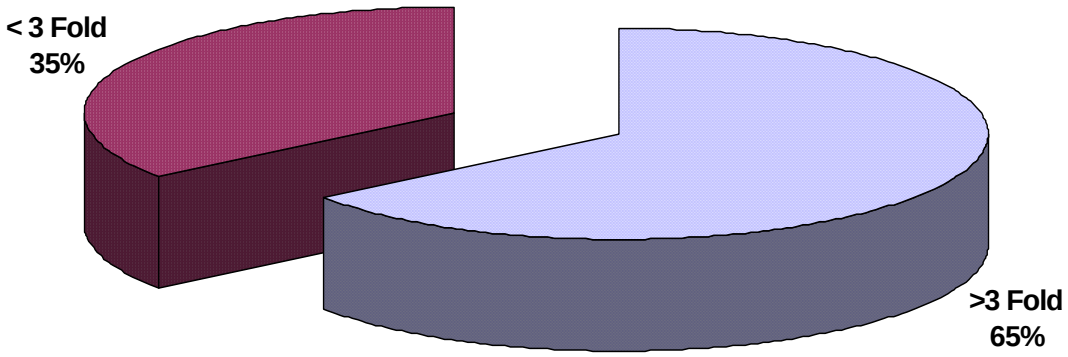
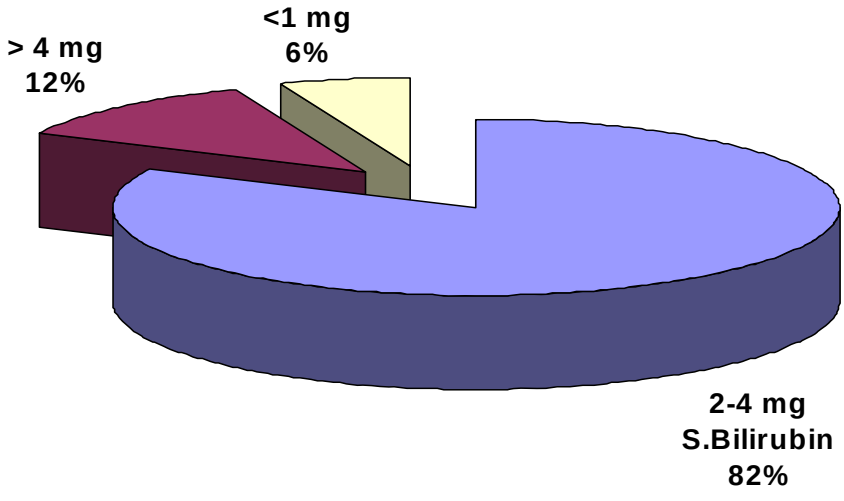


Figure 2.4
LIVER FUNCTION TEST

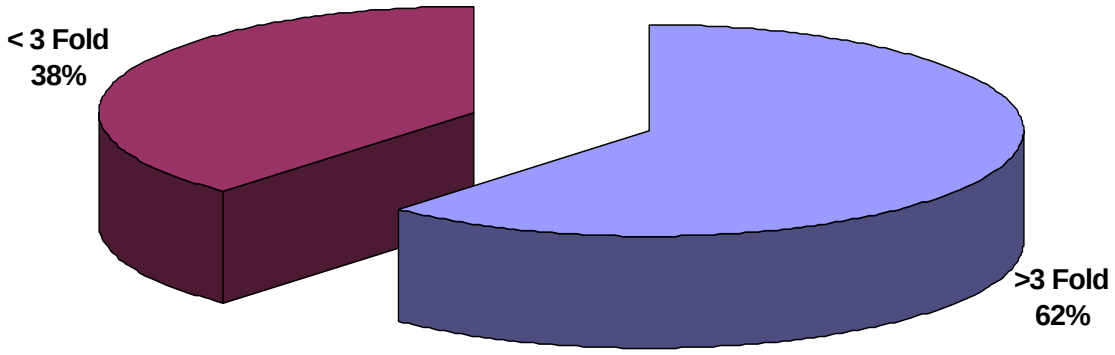
(a)
AST



(b)
SERUM BILIRUBIN



(c)
ALT



(d)
SERUM AMYLASE

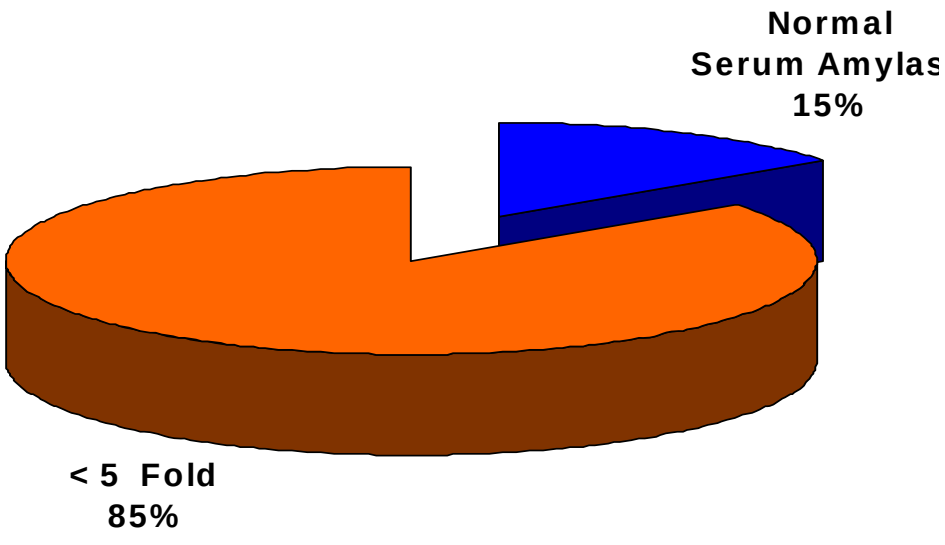
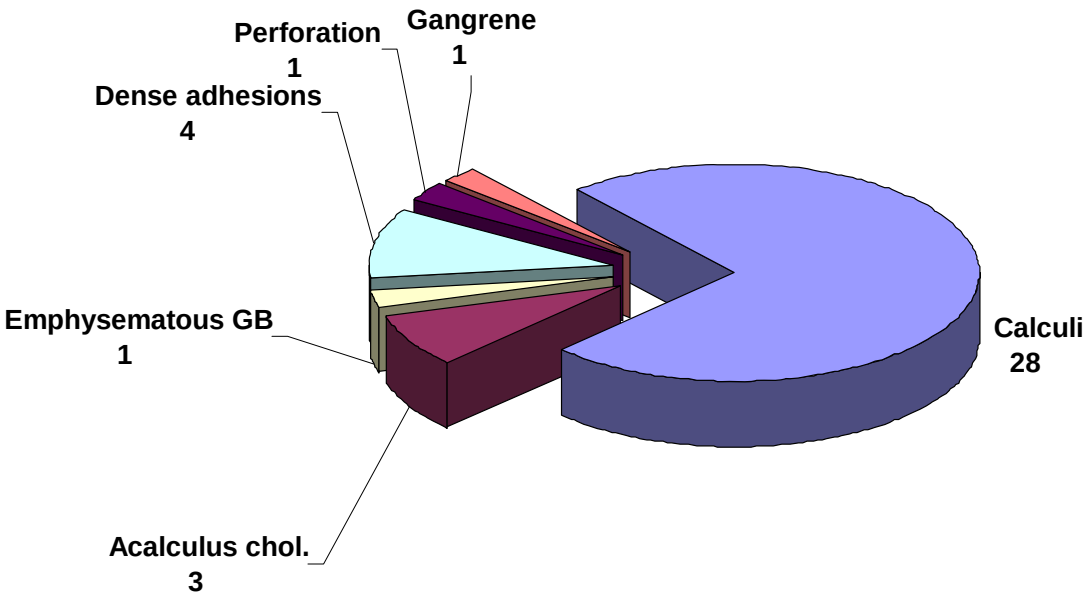


Figure- 2.5

PER-OPERATIVE FINDINGS



BIBLIOGRAPHY

1. Sabiston:Text Book of Surgery - The Biological Basis of Modern Surgical Practice , 17th Edition.
2. Bailey And Love's**:** Short Practice of Surgery,24th Edition.
3. Seymour, I.Schwartz, G.Tom Shires , Frank C.Spencor **:** Principles Of Surgery - 8th Edition.
4. Sleisenger And Fordtran's : Gastrointestinal and Liver Diseases (Vol. 1), 7th Edition.
5. Oxford Text Book Of Surgery**.** *Elsevier* Publications, UK, 1991.
6. Cuschieri: Essential Surgical Practice, 3rd Edition.
7. Henry Gray's**:** Anatomy, 37th Edition.
8. Palanivelu's: Text Book of Laprascopic Surgery , 2nd Edition, 1998.
9. Alexander Mc Gregor's: A Synopsis of Surgical Anatomy, 1998.
10. Bernhoft: Composition And Morphology and Classification of Gall Stones,1988.
11. Farguharsan's: Text Book of Operative Surgery, 9th Edition,2000.
12. Nyhus: Mastery of Surgery , Year 2000 Revised Edition.
13. Harrison's: Principles of Internal Medicine, 16th Edition, 2004.
14. Khuroo M.S., Mahajan K, Zarggar SA *et al:* Prevalence of biliary tract diseases in India. A sinographic stuy in adult population in Kashmir 1992.

15. Jarvinen: The early cholecystectetomy for acute cholecyctitis, Ann. of Surgery, 1982, 191: 501-503.

16. Maingot's, Abdominal Operations, 10th Edition.

17. North American Clinics Of Gastroenterology, 0889-855, 3/94

18. Laing – Ultrasonic evaluation of acute abdomen. Radiology – 1996

19. Robbins**,** Pathological Basis of Disease, 15th Edition

20. Sarin Sk, Negi Vs, Dewan R, *et al.,*: High familial prevalence of gallstones in the first-degree relatives of gallstone patients. *Hepatology* 22:138, 1995.

21. Gracie WA, Ransohoff DF: The natural history of silent gallstones: The innocent gallstone is not a myth. *N. Engl J Med* 307:798, 1982.

22. Valdivieso V, Covarrubias C, Siegel F**,** *et al***:** Pregnancy and cholelithiasis: Pathogenesis and natural course of gallstones diagnosed in early puerperium. *Hepatology* 17:1, 1993.

23. Shaw Sj, Hajnal F, Lebovitz Y, *et al*: Gallbladder dysfunction in diabetes mellitus*. Dig Dis Sci* 38:490, 1993.

24. Zeman RK**.** Gallbladder imaging. The state of art. Gastroenterol Clinics North America 1991; 20 : 127-56.

25. Edlund Y, Olsson O: Acute Cholecystitis: Its aetiology and course, with special reference to the timing of cholecystectomy. Acta Chir Scand, *BMJ*,120:479, 1994.

26. Rathnaswami A, Vijayan J, Omprakash R, Balasubramanian S, Rangabashyam N. Gallstone diseases - our experience. South Indian Journal Clinics 1989; 3 : 89-93.

27. Attili AF, Capocaccia R, Carulli N**,** *et al*: Factors associated with gallstone disease in the MICOL experience: Multicenter Italian Study on Epidemiology of Cholelithiasis. *Hepatology* 26:809, 1997

28. Lujan JA, Parrilla P, Robles R**,** *et al*: Laparoscopic cholecystectomy in the treatment of acute cholecystitis. *J Am Coll Surg* 181:75, 1995.

29. Fletcher Dr, Hobbs MS, Tan P**,** *et al*: Complications of cholecystectomy: Risks of the laparoscopic approach and protective effects of operative cholangiography: A population-based study. *Ann Surg* 229:449–457, 1999.

30. Jayanthi V**,** Pattern of gall stone disease in Chennai city, South India - a hospital based survey. Journ Assoc of physicians of India 1996;44 : 461–4.

31. Tandon RK, Thakur US, Basak AK, Lal K, Jayanthi v, Nijahawan S. Pigment gallstone predominate in south India (abs.) Indian J Gastroenterol 1994; 13 : A18.

32. Sarin SK, Kapur BML, Tandon RK, Cholesterol and pigment gallstones Northern India. A prospective analysis. Dig Dis Sci 1986; 31: 1045 – 5.

33. APPENDIX 1994-1995 SESSIONS. Gazette of Tamil Nadu.

34. The Rome Group for epidemiology and prevention of cholelithiasis (GREPCO). The epidemiology of gallstone disease in Rome, Italy. Part 1. Prevalence data . Hepatology 1988; 9: 904-6.

35. V.Jayanthi , Prasanthi R, Surendran R Palanivelu C et al Epidemiology of gall stone disease. BHJ; 1998.

36. Diehl AK, Schwesinger WH et al : Clinical correlates of gall stone composition ; Am J Gastroenterol 1995;90: 967-72

37. Trotman BW, Soloway RD : Clinical and epidemiological aspects ; Dig.Dis 1975;20:735-9

38. David Lane MD emedicine : Walter Reed Army Medical Center : Mar 2005

39. Robert T Harvey, Wallace Miller : RSNA 1999 : 213: 813-36

40. Ballal M, Jyothi KN, Antony B etal : Bacteriological spectrum of cholecystitis & its antibiogram : Indian J Med Microbiol : 1999.

41. Al-Haijai N, Duca S etal : Incidences & post op complications of lap cholecystectomy : 3 rd surgical clinic ; 3400 cliniq Napola Romania.

MASTER CHART

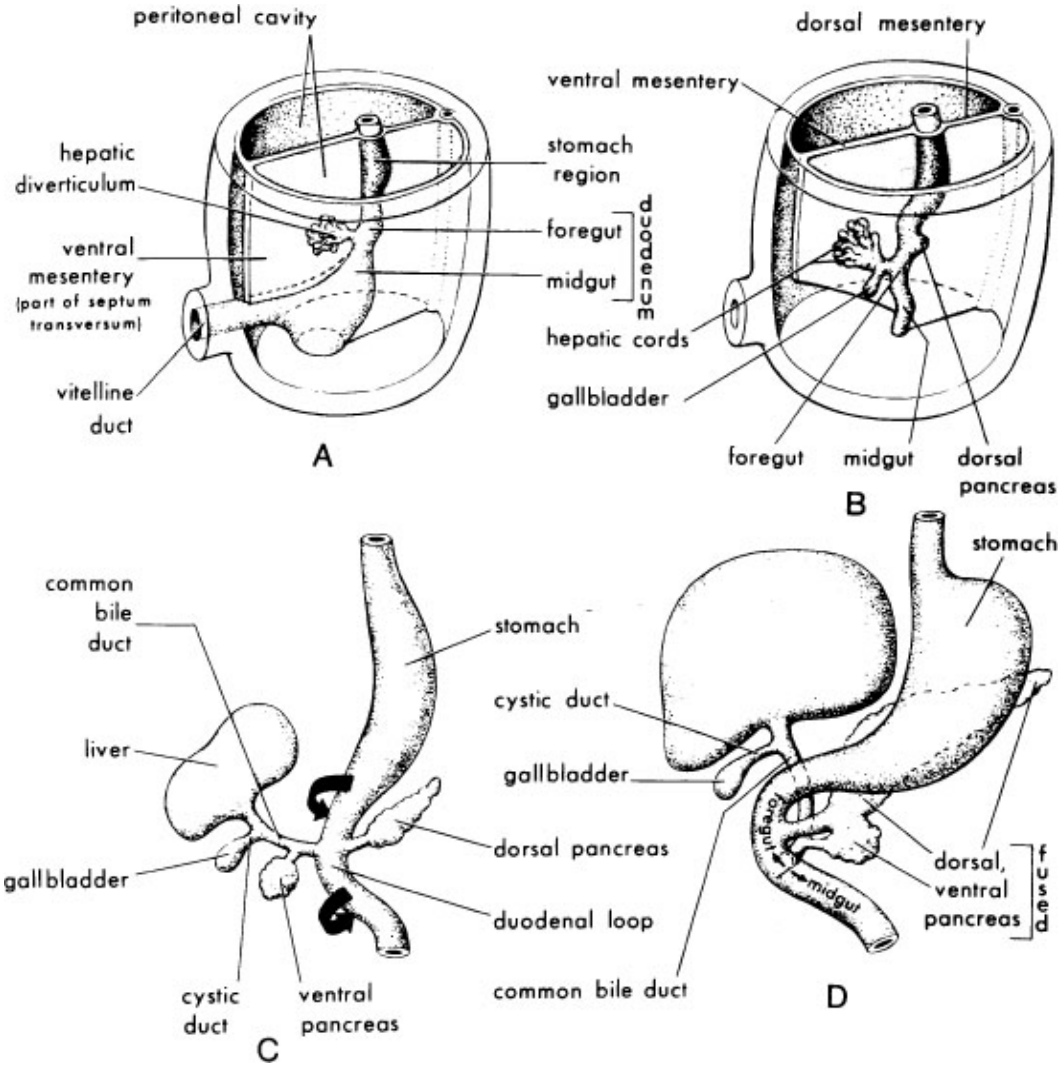
. o.	NAME	AGE YRS	SEX	I.P No.	DIAGNOSIS	INVESTIGATIONS		TREATMENT OFFERED	POST-OPERATIVE PERIOD
						LFT[1]	DIAGNOSTIC IMAGING		
	KASTURI	45	F	297896	CC[2]	NORMAL	USG[4]	**OC[6]**	**UNEVENTFUI**
	JEYALAKSHMI	38	F	297926	CC	NORMAL	USG	**OC**	**UNEVENTFUI**
	MALATHY	43	F	302115	CC	NORMAL	USG	LC[7]	UNEVENTFUL
	SARASWATHI	48	F	303855	CC	NORMAL	USG	OC	COMPLICATED
	AJEERA	14	F	304521	CC	NORMAL	USG	LC	UNEVENTFUL
	SATHISH	37	M	324557	CC	NORMAL	USG	OC	COMPLICATED
	LINKESH	16	M	325872	CC	NORMAL	USG	OC	UNEVENTFUL
	MAHESWARI	58	F	422557	CC	ELEVATED	USG & CT	OC	COMPLICATED
	MARAGATHAM	62	F	425682	AC[3]	NORMAL	USG	OC	COMPLICATED
)	KUMARESWAR	57	M	537789	CC	ELEVATED	USG & CT[5]	OC	COMPLICATED
.	JAFINSULTHAN	46	F	542217	CC	NORMAL	USG	LC	UNEVENTFUL
!	PUSPHA	52	F	557654	CC	NORMAL	USG	OC	UNEVENTFUL
3	PARAMESWARI	56	F	578962	CC	ELEVATED	USG & CT	OC	COMPLICATED
4	PREMALATHA	30	F	478572	CC	NORMAL	USG	LC	UNEVENTFUL
5	RAVANNIAH	51	M	479552	CC	NORMAL	USG	OC	UNEVENTFUL
6	MALLIKA	64	F	487571	AC	NORMAL	USG	CM[8]	NIL RELEVENT
7	KUNDAN SINGH	45	M	578965	CC	ELEVATED	USG & CT	LC	COMPLICATED
8	LEELA	22	F	578411	CC	NORMAL	USG	CM	NIL RELEVANT
)	PARAMASIVAM	54	M	635413	CC	ELEVATED	USG&CT	OC	COMPLICATED
)	VENKATASWAMY	52	M	645874	AC	NORMAL	USG	OC	COMPLICATED
.	SARANYA	25	F	747874	CC	NORMAL	USG	CM	NIL RELEVENT
!	NARMATHA	47	F	754812	CC	NORMAL	USG	LC	UNEVENTFUL
3	SELVAM	38	M	456156	CC	NORMAL	USG	OC	UNEVENTFUL
4	NIRMALA	42	F	557251	CC	ELEVATED	USG & CT	OC	COMPLICATED
5	MANJULA	56	F	447787	CC	NORMAL	USG	OC	UNEVENTFUL
6	MANIMARAN	44	M	323978	CC	NORMAL	USG	LC	UNEVENTFUL
7	MADUBALA	46	F	458624	CC	NORMAL	USG	LC	COMPLICATED
8	SARASWATHI	49	F	489611	CC	NORMAL	USG	OC	UNEVENTFUL
)	PARVATHY	48	F	571246	CC	ELEVATED	USG & CT	CM	NIL RELEVENT
)	ARJUNAN	53	M	584417	AC	NORMAL	USG	OC	COMPLICATED
.	THENMOZHI	39	F	548874	CC	NORMAL	USG	LC	COMPLICATED
!	INDIRA	31	F	487578	CC	NORMAL	USG	LC	UNEVENTFUL
3	GOMATHY	44	F	324781	CC	NORMAL	USG	OC	UNEVENTFUL
4	MANIMEGALAI	37	F	457512	CC	NORMAL	USG	OC	UNEVENTFUL

1. LFT – Liver function tests 2. CC – Calculus cholecystitis 3. AC – Acalculus cholecystitis 4. USG – Ultrasonogram

5. CT - Computed tomography 6. OC – Open cholecystectomy 7. LC – Laparoscopic cholecystectomy

8. CM – Conservative management

STAGES IN DEVELOPMENT OF LIVER / GALL BLADDER/ EXTRAHEPATIC BILIARY APPARATUS / PANCREAS & DUODENUM



A – 4 WEEKS B&C – 6 WEEKS D – 8 WEEKS

Fig.1.1

ANATOMY OF EXTRA HEPATIC BILIARY APPARATUS AND CHOLEDOCHODUODENAL JUNCTION

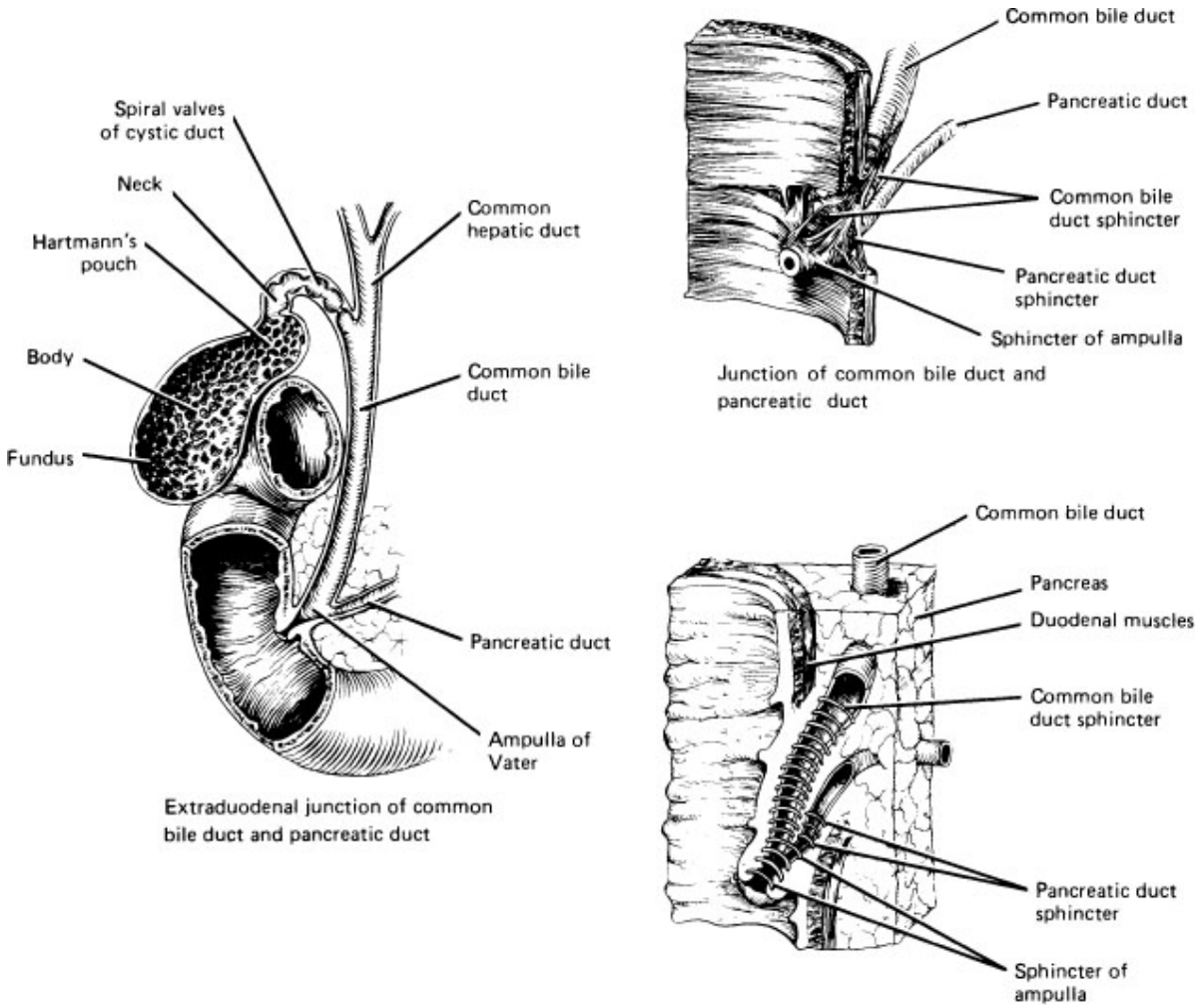


Fig. 1.2

PARTS OF ENTROHEPATIC CIRCULATION

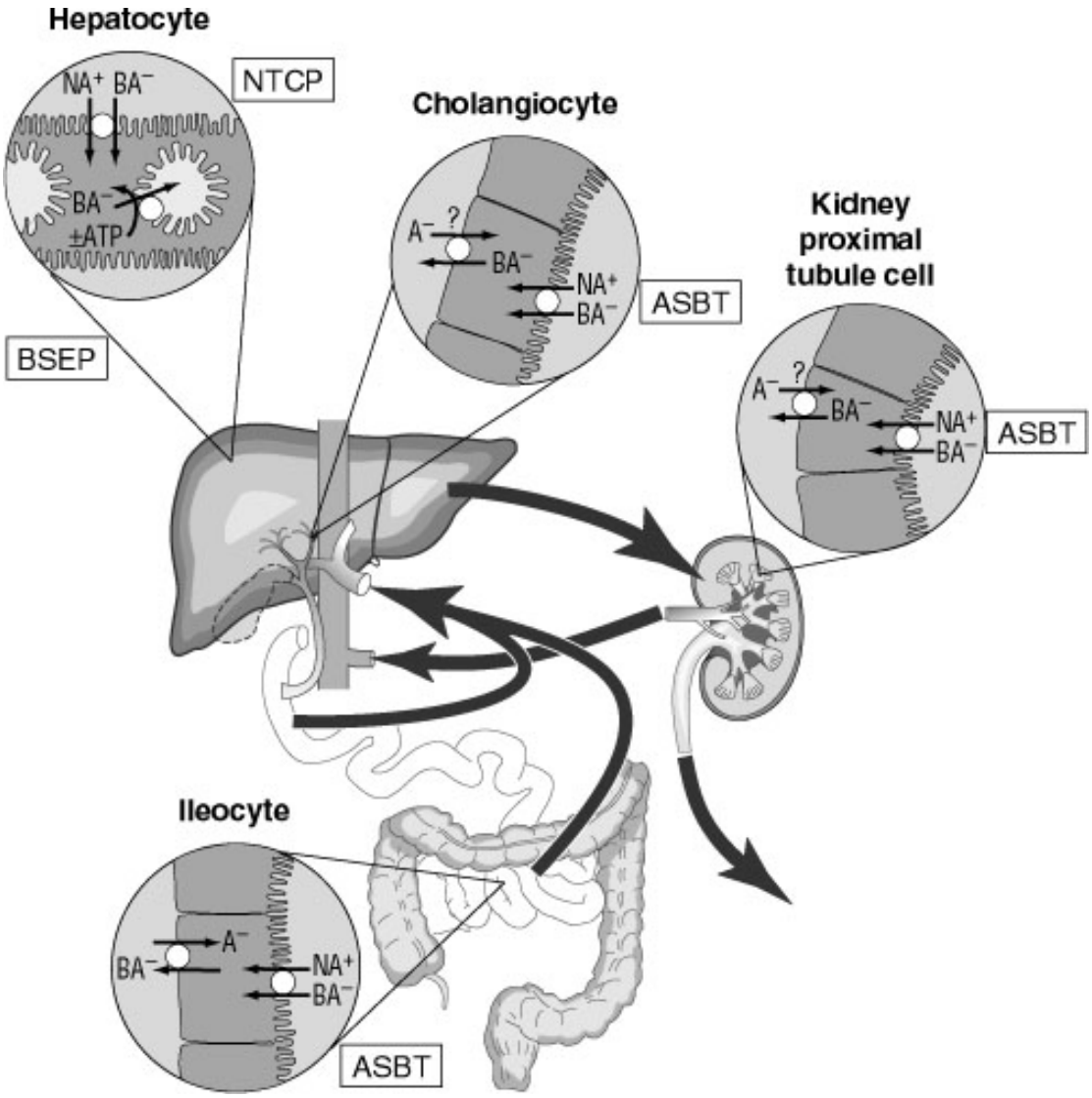


Fig.1.3

SCHEME FOR BROWN PIGMENT STONE FORMATION

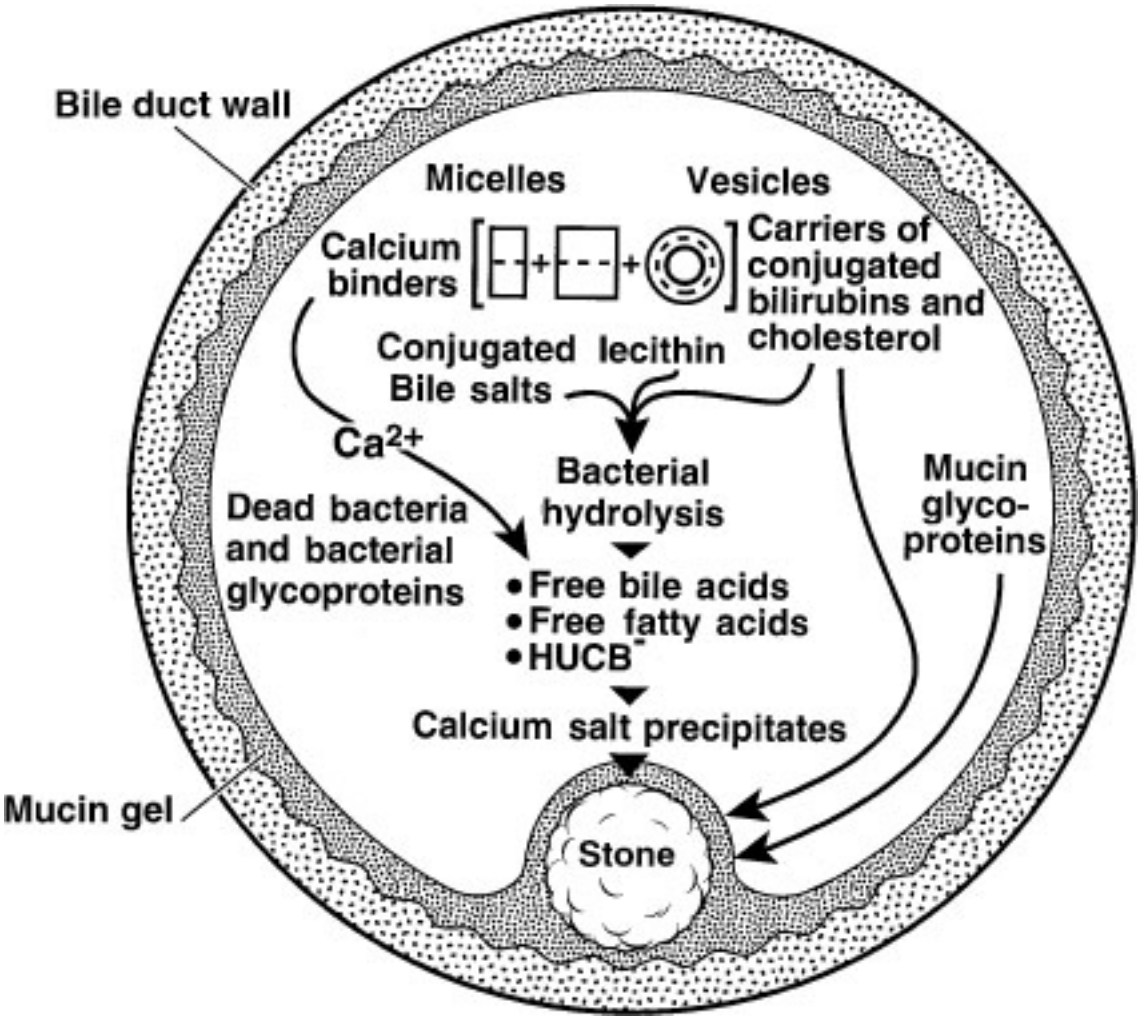


Fig. 1.4

CLINICAL MANIFESTATIONS

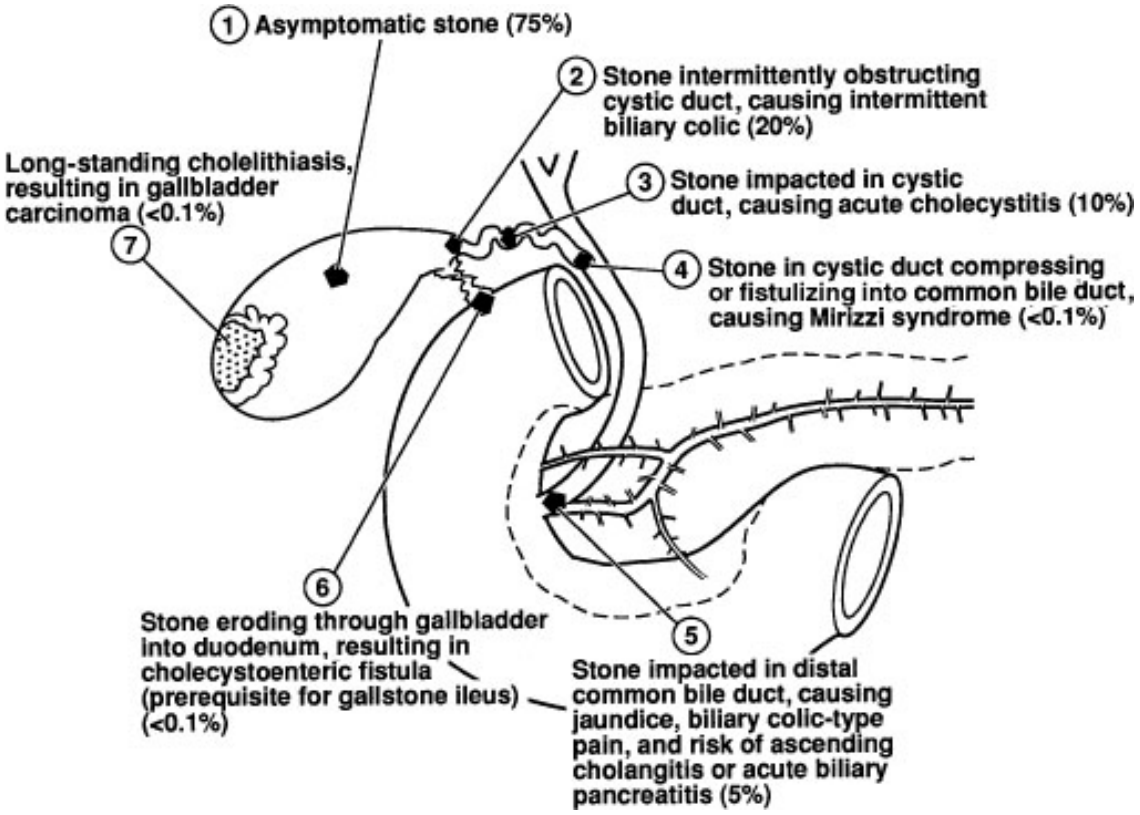


Fig.1. 5

COMMON CLINICAL MANIFESTATIONS OF GALL STONE DISEASE

TABLE - a

	BILIARY COLIC	ACUTE CHOLECYSTITIS	CHOLEDOCHO-LITHIASIS	CHOLANGITIS
Pathophysiologic condition	Intermittent obstruction of cystic duct No inflammation of gallbladder mucosa	Impacted stone in cystic duct Acute inflammation of gallbladder mucosa Secondary bacterial infection in ≈50%	Intermittent obstruction of CBD	Impacted stone in CBD causing bile stasis Bacterial superinfection of stagnant bile Early bacteremia
Symptoms	Severe, poorly localized epigastric or RUQ visceral pain growing in intensity over 15 min and remaining constant for 1–6 hr, often with nausea Frequency of attacks varies from days to months Gas, bloating, flatulence, dyspepsia *not* related to stones	75% Preceded by attacks of biliary colic Visceral epigastric pain gives way to moderately severe, localized pain in RUQ, back, shoulder, or (rarely) chest Nausea with some emesis-frequent Pain lasting >6 hr suggests cholecystitis (vs. colic)	Often asymptomatic Symptoms (when present) indistinguishable from biliary colic symptoms Predisposes to cholangitis and acute pancreatitis	Charcot's triad (pain, jaundice, fever) present in 70% May be mild, transient pain often accompanied by chills Mental confusion, lethargy, and delirium suggestive of bacteremia
Physical findings	Mild to moderate gallbladder tenderness during attack with mild residual tenderness lasting days Often completely normal examination result	Febrile, usually <102°F unless complicated by gangrene or perforation Right subcostal tenderness with inspiratory arrest (Murphy's sign) Palpable gallbladder in 33%, especially in first attack Mild jaundice in 20%, higher frequency in elderly	Often completely normal examination result if obstruction intermittent Jaundice with pain suggestive of stones; painless jaundice and palpable gallbladder suggestive of malignancy	Fever in 95% RUQ tenderness in 90% Jaundice in 80% Peritoneal signs in 15% Hypotension with mental confusion in 15% suggestive of gram-negative sepsis
Laboratory findings	Usually normal In patients with findings of only uncomplicated biliary colic, elevated bilirubin, alkaline phosphatase, or amylase level suggestive of coexisting CBD stones	Leukocytosis of 12,000–15,000/mm³ with left shift common Serum bilirubin may be 2–4 mg/dL and aminotransferase and alkaline phosphatase levels may be elevated, even in absence of CBD stone or hepatic infection Mild serum amylase elevation even in absence of pancreatitis If bilirubin >4 mg/dL or amylase >1000 U/L suspect CBD stone	Elevated serum bilirubin and alkaline phosphatase levels seen with CBD obstruction Serum bilirubin level >10 mg/dL suggestive of malignant obstruction or coexisting hemolysis Transient spike in serum aminotransferase or amylase levels suggestive of stone passage	Leukocytosis in 80%; normal WBC count with left shift may be only hematologic finding in 20% Serum bilirubin level >2 mg/dL in 80%, but when <2 mg/dL diagnosis may be missed Serum alkaline phosphatase level usually elevated Blood culture results usually positive, especially during chills or fever spike; grow two organisms in one half of patients
Diagnostic tests	Ultrasonography OCG Meltzer-Lyon test	Ultrasonography Hepatobiliary scintigraphy (DISIDA, HIDA scans) Abdominal CT	ERCP THC	ERCP THC
Natural history	After initial attack, no further symptoms in 30% In remainder development of symptoms at rate of 6%/year and of severe complications at rate of 1%/year	Spontaneous resolution in 50% in 7–10 days without surgery Untreated, 10% complicated by localized perforation, 1% by free perforation and peritonitis	Natural history not well defined, but complications more frequent and severe than for asymptomatic (gallbladder) stones	High mortality rate if unrecognized with death from septicemia Dramatic improvement of survival rate with emergent decompression of CBD (usually by ERCP)
Treatmer	Elective laparoscopic cholecystectomy with IOC ERCP for stone removal if stones on IOC	Cholecystectomy with IOC If stones on IOC, then CBD exploration or ERCP for stone removal	Stone removal at time of ERCP followed by early laparoscopic cholecystectomy	Emergency ERCP with stone removal or at least biliary decompression Antibiotics to cover gram-negative organisms Interval cholecystectomy

UNCOMMON COMPLICATIONS OF GALLSTONE DISEASE

TABLE - b

COMPLICATION	PATHOGENESIS	CLINICAL MANIFESTATIONS	DIAGNOSIS/TREATMENT
Emphysematous cholecystitis	Secondary infection of gallbladder wall with gas-forming organisms (*Clostridium welchii, Escherichia coli,* anaerobic streptococci) More common in elderly and diabetic men; often occurs in absence of stones (see Chap. 58)	Similar symptoms and signs to acute cholecystitis but more toxic presentation	Plain abdominal series that may show gallbladder fossa gas Ultrasonography and CT sensitive for confirming gas Treatment with intravenous antibiotics including anaerobic coverage and early cholecystectomy Morbidity and mortality rates high
Cholecystoenteric fistula	Erosion of (usually large) stone through gallbladder wall into adjacent bowel, most often duodenum, and less often hepatic flexure, stomach, and jejunum	Similar symptoms and signs to acute cholecystitis; fistula potentially clinically silent Lingering symptoms that suggest persistence of gallbladder stones Stones >25 mm, especially in elderly women, that may produce bowel obstruction or "gallstone ileus"; terminal ileum most frequent site of obstruction	Plain abdominal series that may show biliary tree gas and SBO in gallstone ileus Contrast GI series that may demonstrate fistula Fistula from solitary stones that pass that may close spontaneously Cholecystectomy and bowel closure curative Gallstone ileus requiring emergency laparotomy; diagnosis often delayed; resulting mortality rate ≈20%
Mirizzi's syndrome	Impacted stone in gallbladder neck or cystic duct with extrinsic compression of CBD by accompanying inflammation	Jaundice and RUQ pain	ERCP that demonstrates extrinsic CBD compression Preoperative diagnosis important to guide surgical approach and minimize CBD injury risk
Porcelain gallbladder	Intramural calcification of gallbladder wall, usually associated with stones	No symptoms attributable to calcified wall, but gallbladder carcinoma late complication in ≈20% (see Chap. 60)	Plain abdominal series or CT that shows intramural gallbladder wall calcification Prophylactic cholecystectomy indicated to prevent carcinoma

CBD, common bile duct; CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; GI, gastrointestinal; RUQ, right upper quadrant; SBO, small bowel obstruction

OVERVIEW OF INVESTIGATIONS FOR BILIARY TRACT DISEASES

Table - c

TECHNIQUE	CONDITION TESTED FOR	FINDINGS/COMMENTS
Ultrasonography	Cholelithiasis	Stones that appear as mobile, dependent echogenic foci in gallbladder lumen with acoustic shadowing Sludge that appears as layering echogenic material without shadows Sensitivity >95% for stones >2 mm Specificity >95% for stones with acoustic shadows Rarely stone-filled gallbladder contracted and difficult to see with "wall-echo-shadow" sign Best single test for stones in gallbladder
	Choledocholithiasis	Stones in CBD only seen sonographically in ≈50% of cases; can be inferred by finding of dilated CBD (>6 mm diameter) in ≈75%; ultrasonography able to confirm but not exclude CBD stones
	Acute cholecystitis	Sonographic Murphy's sign (focal gallbladder tenderness under the transducer) has positive predictive value >90% for detecting acute cholecystitis when stones seen Pericholecystic fluid (in absence of ascites) and gallbladder wall thickening to >4 mm (in absence of hypoalbuminemia) nonspecific findings that suggest acute cholecystitis
EUS	Choledocholithiasis	Highly accurate for excluding or confirming stones in CBD Sensitivity of 93%, specificity of 97% Concordance of EUS and ERCP diagnoses of 95% EUS (vs. ERCP) used by experienced operators in excluding CBD stones
OCG	Cholelithiasis	Stones that appear as mobile filling defects in opacified gallbladder Sensitivity and specificity >90% when gallbladder opacified; nonvisualization in 25% of tests that may be due to many causes other than stones Opacification of gallbladder that demonstrates patency of cystic duct (prerequisite for medical dissolution therapy or lithotripsy) Potential use in evaluation of acalculous gallbladder diseases (e.g., cholesterolosis, adenomyomatosis) (see Chap. 58)
Cholescintigraphy (hepatobiliary scintigraphy, HIDA, DISIDA scans)	Acute cholecystitis	Assessment of cystic duct patency On normal scan radioactivity in gallbladder, CBD, and small bowel in 30–60 min Positive scan defined as nonvisualization of gallbladder with preserved excretion into CBD or small bowel Sensitivity ≈95%, specificity ≈90%, with false-positive results in fasting, critically ill patients With CCK stimulation, gallbladder "ejection fraction" determined and may help evaluate patients with acalculous biliary pain (see Chap. 58) *Normal scan result virtually excludes acute cholecystitis*
ERCP	Cholelithiasis	With contrast medium flow retrograde into gallbladder, stones that appear as filling defects detectable with sensitivity of ≈80%; ultrasonography mainstay to confirm cholelithiasis
	Choledocholithiasis	ERCP gold standard test for stones in CBD, with sensitivity and specificity rates ≈95% Ability to extract stones (or drain infected bile) lifesaving in severe cholangitis, reducing need for CBD exploration
CT/MRI	Complications	Not well suited for detecting uncomplicated stones; standard CT excellent test for detecting complications (e.g., abscess formation, gallbladder perforation, or CBD stone) or pancreatitis Spiral CT and MR cholangiography potentially useful as noninvasive means of excluding CBD stones

CBD, common bile duct; CCK, cholecystokinin; CT, computed tomography; DISIDA, diisopropyl iminodiacetic acid; ERCP, endoscopic retrograde cholangiopancreatography; EUS, endoscopic ultrasonography; HIDA, hydroxy iminodiacetic acid; MRI, magnetic resonance imaging; OCG, oral cholecystography.

DIAGNOSTIC CRITERIA

Table d

TECHNIQUE	FINDINGS
Clinical examination	Right upper quadrant tenderness and Murphy's sign are helpful if present but are lacking in three fourths of cases Unexplained fever, leukocytosis, or hyperamylasemia are frequently the only signs
Ultrasonography	Thickened gallbladder wall, defined as >4 mm thickness in the absence of ascites or hypoalbuminemia (<3.2 g/dL) Sonographic Murphy's sign, defined as maximum tenderness over the ultrasonographically localized gallbladder Pericholecystic fluid collection • *Bedside availability is major advantage*
Computed tomography	Thickened gallbladder wall, defined as >4 mm thickness in the absence of ascites or hypoalbuminemia (<3.2 g/dL) Pericholecystic fluid, subserosal edema (in the absence of ascites), intramural gas, or sloughed mucosa • *Best test for excluding other intra-abdominal pathology but requires moving patient to scanner*
Hepatobiliary scintigraphy	Nonvisualization of the gallbladder with normal excretion of dye into the bile duct and duodenum is defined as a positive test for acute cholecystitis Critically ill immobilized patients may have false-positive scans because of viscus bile Morphine augmentation may reduce the number of false-positive results • *Better at excluding acute cholecystitis than confirming it*

VENN DIAGRAM SHOWS 3 IMPORTANT FACTORS ARE REQUIRED

FOR GALL STONE FROMATION

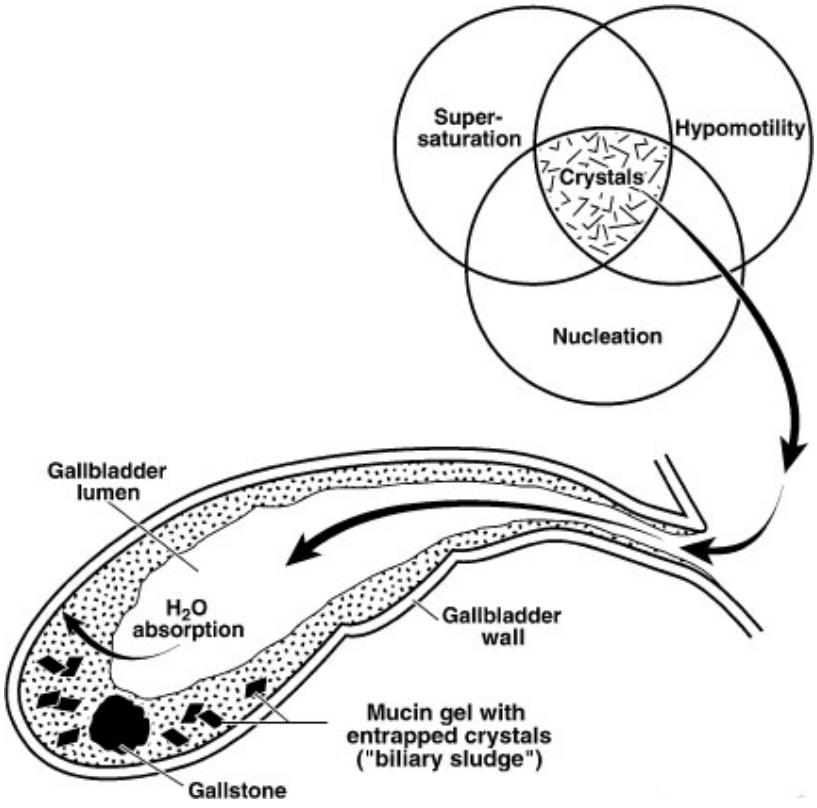


Fig.1. 6

PROFORMA

NAME: AGE: SEX:
D.O.A.: D.O.D: I.P.No.:
PROVISIONAL DIAGNOSIS:
FINAL DIAGNOSIS :
TREATMENT :

PRESENT HISTORY:

►PAIN - DURATION/ LOCATION/ CHARACTER/RADATION
AGGRAVATING AND RELIVING FACTORS
►JAUNDICE
► NAUSEAVOMITING
►FEVER
►DYSPEPSIA

PAST HISTORY :

SIMILAR COMPLAINTS/ GALL STONES:
DIABETES:
JAUNDICE: PREVIOUS SURGERY:
HYPERTENSION: RESPIRATORY DISEASE:

PERSONAL HISTORY:

MARITAL STATUS: SOCIOECONOMIC STATUS:
OBESITY/ BUILT: DIET : VEG/NONVEG
SMOKING/ ALCOHOL: DRUG INTAKE:
BOWEL & BLADDER HABITS:

FAMILY HISTORY :

SIMILAR ILLNESS:
DIABETES/HYPERTENSION:
STONE DISEASE :
OBESITY:

GENERAL EXAMINATION :

ANAEMIA : JANUDICE : HYDRATION :
PULSE : B.P.: RESPIRATORY RATE: TEMP.:
HEART AND LUNGS :

EXAMINATION OF ABDOMEN:

TENDERNESS – RT. HYPOCHONDRIUM
LIVER/ GALL BLADDER/ SPLEEN:
FREE FLUID:
GUARDING / RIGIDITY/ REBOUND TENDERNESS/ MURPHYS SIGN

INVESTIGATIONS:

URINE – BILE SALTS/ PIGMENTS BLOOD- HB%,TC,DC,ESR
LIVER FUNCTION TESTS: SERUM CHOLESTEROL:
SERUM AMYLASE: ECG/ BLOOD- UREA/ SUGAR
PLAIN X RAY : ABD./ CHEST ULTRASOUND ABDOMEN:
CT SCAN ABDOMEN:
ERCP

TREATMENT :

SURGERY : OPEN/ LAPAROSCOPIC
CONSERVATIVE :

POST-OPERATIVE PERIOD:

FEVER/ SEPSIS/ WOUND INFECTION/ RESIDUAL STONE /

FISTULA/ABDOMINAL COLLECTION

POST OP INVESTIGATIONS:

1. BILE CULTURE
2. STONE ANALYSIS

PROFORMA

NAME: AGE: SEX:
D.O.A.: D.O.D: I.P.No.:
PROVISIONAL DIAGNOSIS:
FINAL DIAGNOSIS :
TREATMENT :

PRESENT HISTORY:

►PAIN - DURATION/ LOCATION/ CHARACTER/RADATION
AGGRAVATING AND RELIVING FACTORS
►JAUNDICE
► NAUSEAVOMITING
►FEVER
►DYSPEPSIA

PAST HISTORY :

SIMILAR COMPLAINTS/ GALL STONES:
DIABETES:
JAUNDICE: PREVIOUS SURGERY:
HYPERTENSION: RESPIRATORY DISEASE:

PERSONAL HISTORY:

MARITAL STATUS: SOCIOECONOMIC STATUS:
OBESITY/ BUILT: DIET : VEG/NONVEG
SMOKING/ ALCOHOL: DRUG INTAKE:
BOWEL & BLADDER HABITS:

FAMILY HISTORY :

SIMILAR ILLNESS:
DIABETES/HYPERTENSION:
STONE DISEASE :
OBESITY:

GENERAL EXAMINATION :

ANAEMIA : JANUDICE : HYDRATION :
PULSE : B.P.: RESPIRATORY RATE: TEMP.:
HEART AND LUNGS :

EXAMINATION OF ABDOMEN:

TENDERNESS – RT. HYPOCHONDRIUM
LIVER/ GALL BLADDER/ SPLEEN:
FREE FLUID:
GUARDING / RIGIDITY/ REBOUND TENDERNESS/ MURPHYS SIGN

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URINE – BILE SALTS/ PIGMENTS BLOOD- HB%,TC,DC,ESR
LIVER FUNCTION TESTS: SERUM CHOLESTEROL:
SERUM AMYLASE: ECG/ BLOOD- UREA/ SUGAR
PLAIN X RAY : ABD./ CHEST ULTRASOUND ABDOMEN:
CT SCAN ABDOMEN:
ERCP

TREATMENT :

SURGERY : OPEN/ LAPAROSCOPIC
CONSERVATIVE :

POST-OPERATIVE PERIOD:

FEVER/ SEPSIS/ WOUND INFECTION/ RESIDUAL STONE /

FISTULA/ABDOMINAL COLLECTION

POST OP INVESTIGATIONS:

1. BILE CULTURE
2. STONE ANALYSIS